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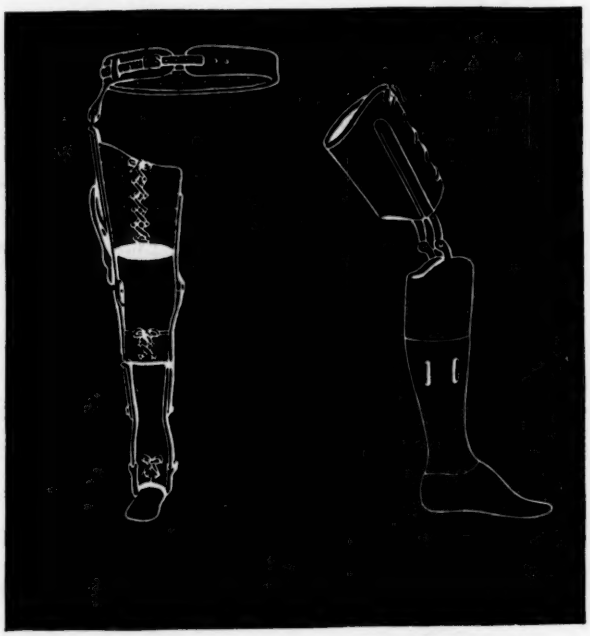

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NEW ENGLAND SURGICAL SOCIETY

CERTAIN ASPECTS OF HAND SURGERY*

BY TORR WAGNER HARMER, M.D.†

THE objective in repair and reconstruction surgery of the hand is restoration of function. In infections of the hand the objectives are drainage and preservation of function. In reconstruction work (tendon grafting, etc.) surgeons are prone to use splints, lest active motions undo their work. In serious infections, as with the hemolytic streptococcus, many of us, when recovery is in sight, are prone to sit back with a grateful sigh. This is the very time we should be instituting special exercises in active motion to preserve function. The bête noir of all tendon work is anchoring adhesions. The best means to combat anchoring adhesions are minimization of operative trauma and early active motions. Operative trauma may be minimized by preoperative diagnosis, adequate exposure, and deliberate and gentle technic. Early active motions following repair work necessitate a method of tendon suture which will not pull out or stretch. I shall show in a short movie later a stitch which I have used with satisfaction since 1909.

In surgery of the hand, be it for immediate repair, acute infection or late reconstruction, the aftercare is quite as important as the operation. Good results cannot be secured without patience and some ingenuity in guiding the convalescence.

I have adopted two general principles in aftercare: one is the principle of immediate active motion without splinting; the other is the principle of immediate active motion with limitation of motion of antagonistic muscles[1].

By immediate active motion is meant the purposeful active use of each of the repaired parts within forty-eight hours of the operation. Each exercise is carried out several times every hour. The early dressings insure sufficient immobilization. This principle of immediate active motion without splinting is applicable in the following situations:—flexors of the fingers in any of the phalanges and in the palm, long flexor of the thumb, flexors or extensors at the wrist without coincident major nerve repair, and extensors of the fingers on the dorsum of the hand and in the first two phalanges. In case of the wrist without coincident major nerve repair a dressing which includes the hand gives sufficient immobilization. In situations other than these the principle of immediate active motion with limitation of antagonistic muscles is used; namely, flexors at wrist with coincident nerve repair, extensors of thumb, and extensors of the fingers at the knuckles and at the terminal phalanges. In the films I shall show devices for limiting antagonistic muscles which, however, permit active motions of the repaired tendons.

The repair of a lacerated wound, even an extensive wound of the anterior surface of the wrist, should not be an exploratory operation. After glancing at the situation of the wound, we should have the patient attempt to repeat with us various motions of the fingers and thumb to test the integrity of the tendons and the nerves. We should have a good idea what our repair problem is before operation is begun. In the next short film is shown a method of testing the extent of the tendon and nerve injury in case of a lacerated wound on the anterior aspect of the wrist,—flexor longus pollicis, flexor brevis—profundus and sublimis of each finger,—lumbricals and interossei—tests for ulna nerve (abduction and adduction of the fingers, abductor minimi digiti—abductor indicis—adductor pollicis)—test for median nerve (apposition of thumb to fingers). The motions of thumb and fingers in the first part of the film also illustrate the method of active motions used after repair,—each phalanx of each digit purposefully several times every hour.

A tendon stitch which I have used satisfactorily since 1909 is now illustrated[2]. (See fig. 1.) It is of No. 7 black silk, once-boiled. It is a simple overcasting affair starting on a tendon border about the width of the tendon from its severed surface. Where the stitch is started a single knot is tied. Several overcastings are then made down to the severed surface. The other margin of the same tendon-end is similarly supplied with a stitch. The distal end of the same tendon is then similarly provided with stitches. This illustrates an important advantage of the stitch. As the tendon-ends are identified at operation they can be provided with stitches and laid aside. When the deepest tendons have

*Read before Annual Meeting of New England Surgical Society at Manchester, N. H., September 28, 1935.

†Harmer, Torr Wagner—Assistant Visiting Surgeon, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 653.

1. Harmer: Boston M. & S. J. **194**: 739, 1926.

2. Boston M. & S. J. **177**: 808, 1917.

been so treated, tying together of the tendons may be done from within out. This method minimizes operative trauma. Again the stitch does not penetrate the core of the tendon at the severed surface. Core-penetrating sutures provoke more scar tissue between the tendon-ends. The scar may stretch producing a little slack which defeats complete restoration of function.

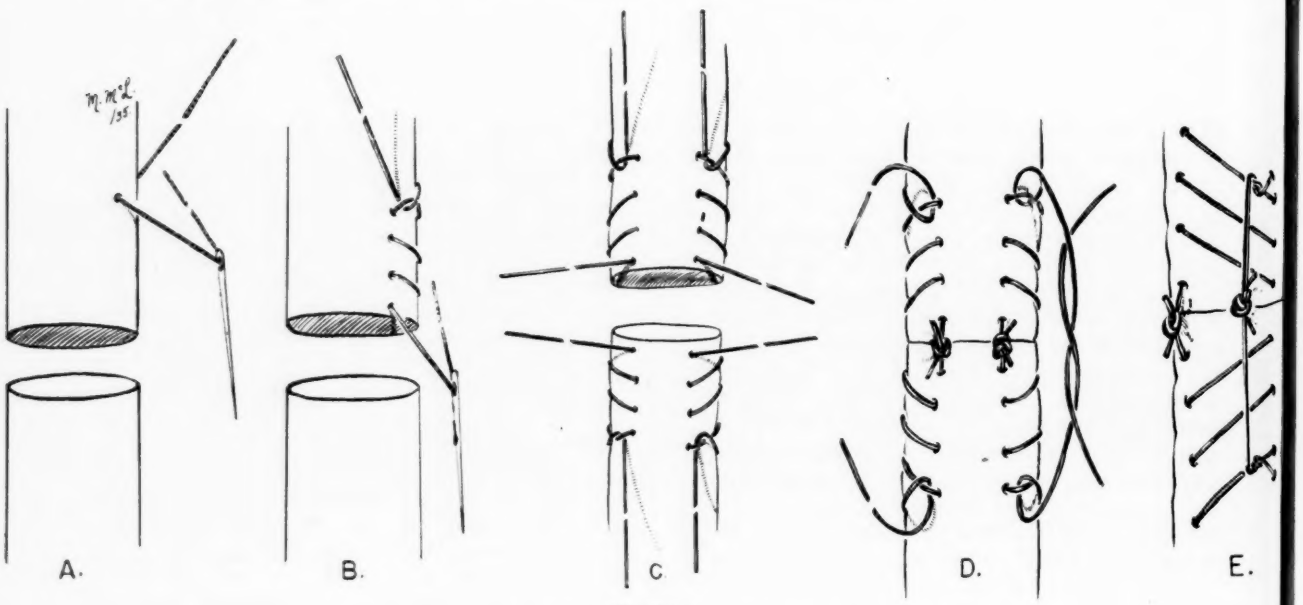


FIGURE 1.

Tendon suture. The size of the silk has been exaggerated. **A.** Start of stitch from under surface of tendon. **B.** Single knot and overcasting down to severed surface which is not penetrated. **C.** Both ends of severed tendon provided with stitches ready to tie. **D.** Shorter stitch ends tied and longer ends being tied. **E.** Side view of tendon with stitches tied. The upper surface of the tendon is to the left.

A few years ago Koch, Mason, and Shearon pointed out the disadvantages of sutures which pierce the core of the tendon end[3, 4].

The film will next illustrate devices for limiting antagonizing muscles; first, in the repair of flexors of the wrist with coincident nerve repair[5]. The film will show an aluminum splint covered with felt and provided with three buckle straps which encircle the forearm, wrist and palm. (See fig. 2.) The splint maintains the wrist in flexion but permits active use of the fingers. Each phalanx is exercised separately as shown previously in the film. Tension on the sutured nerves, which would inhibit their regeneration, is however prevented by the flexed splint.

With the hand in repose the thumb is somewhat flexed. The flexors are stronger than the extensors. Following repair of the extensors, unless this steady pull is relieved, the repaired tendons will be stretched and complete restoration of function will not be attained. This may not be important in a laborer but may be an unsurmountable disability in a middle-aged pianist or harpist. We should always strive for maximum results. I use a banjo splint with a rubber-band to support the antagonizing flexor. The elastic is removed every hour for active exercise of the repaired extensors. The device is also used following the repair of extensor tendons at the knuckles.

3. Koch, S. L., and Mason, M. L.: Surg. Gynec. & Obst. **56**: 1, 1933.
4. Mason, M. L., and Shearon, C. G.: Arch. Surg. **25**: 615, 1932.
5. Harmer, T. W.: S. Clin. North America (June) P. 822, 1921.

The film will show a patient to illustrate the use of the banjo splint with rubber-band support. A fishmonger of eighteen had severed the extensor pollicis longus of his right hand with a fish knife a few hours before I saw him (February 14, 1935). He presented a ¾ inch transverse incised wound over the metacarpophalangeal joint which severed the long extensor of the thumb and opened the joint. Immediate tenorrhaphy was done through a 2½" posterolateral incision. The tendon-ends were found separated about an inch. Few interrupted 00 plain gut to fascia, silk to skin, alcohol dressing, banjo splint with terminal phalanx held in hyperextension by a rubber-band. February 16 active motions started. February 18 discharged from hospital. February 20 (six days after injury) film taken showing patient actively exercising the sutured tendon. March 12 discharged to Insurance Clinic. March 18 resumed former work at former pay.

Following the repair of the thumb extensors the banjo splint with rubber-band is worn for twelve or thirteen days. It is then replaced by a figure-of-eight adhesive strapping to support the proximal joint and a metal splint to support the distal joint in slight hyperextension. The metal splint is of aluminum and is removed each hour for active exercise of the sutured extensors. The strapping is changed once during the next ten days, when it is omitted. The metal splint is worn for another week or

ten days. It may be worn at night for a longer period. (See fig. 3.)

The film will show a patient to illustrate the use of the removable aluminum finger splint. A dentist of thirty-two, on January 10, 1935, severed the long extensor of his left thumb with a saw. Immediate repair by a local physician was attempted but failed. I saw the patient on January 23, found the long extensor functionless and the wound still unhealed. February 1 incision along the radial border of the thumb and first metacarpal exposed the divided tendon bridged by a scar tissue band which was adherent to the surrounding tissues. Tenorrhaphy,

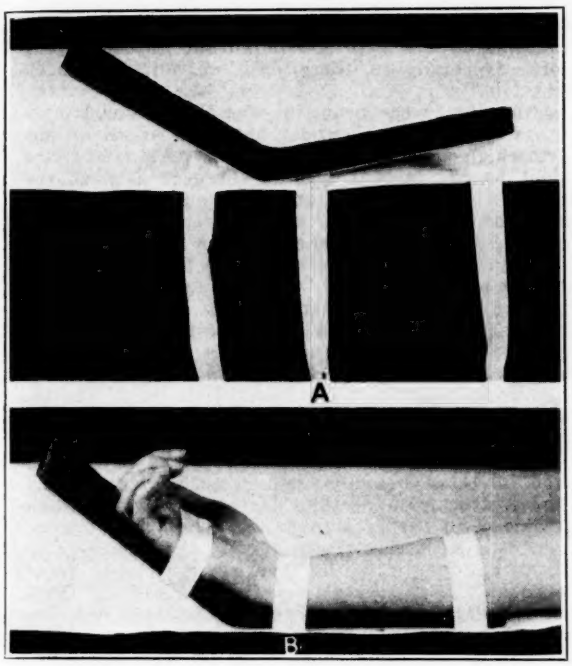
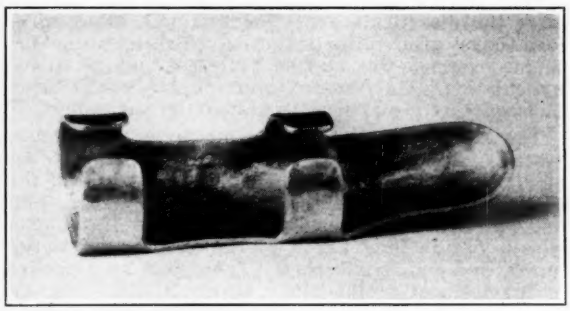


FIGURE 2.

Felt-covered aluminum splint used following repair of nerves and flexor tendons at wrist. **A.** The splint has been gently angulated to receive the flexed wrist. **B.** Splint in use. Although thumb and fingers can be actively moved, extension of the wrist is prevented. The splint does not fit the subject, the forearm portion is too short and the hand portion too long.

closure, dressing and banjo splint, as in previous case. February 3 discharged from hospital. Active exercises as in previous case. February 13, banjo splint removed; figure-of-eight adhesive dressing applied to support proximal joint and removable metal splint to support distal joint. Distal joint to be actively exercised several times every hour. February 23, strapping removed. Use of metal splint continued till March 8. Obtained a perfect result.

The remainder of the film shows several stages in the treatment of two cases desperately ill with hemolytic streptococcus infections. Its purpose is to illustrate three principles in the treatment of such cases: 1, immune transfusion; 2, the feasibility and desirability of early active motions; 3, the preservation of tendons following the drainage of tendon sheaths and bursae. Theoretically in immune transfusion the strain of hemolytic streptococcus from which the donor has recovered and that with which the patient is infected should be identical. Practically, in my experience, absolute specificity is not always necessary. If the bloods are compatible, even if the strain of hemolytic streptococcus is undetermined in either individual, it is intelligent practice to use the recently recovered individual as a donor for a whole blood transfusion to the patient without delaying the transfusion until it is determined that the proposed donor has been and the recipient is infected with the same strain of hemolytic streptococcus. Hemologic studies may be started at the time which may prove profitable, if the transfusion fails. The donor of the second transfusion to the first patient illustrated in the film was known to have recovered from a different



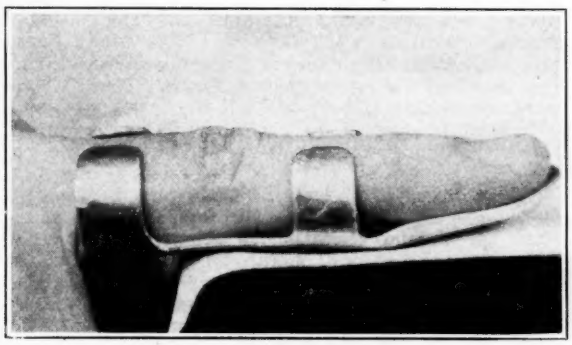


FIGURE 3.

Readily removable aluminum splint used to hold terminal phalanx in extension (to eliminate the antagonistic pull of flexors) following repair of extensors of thumb (as described in text) and avulsions of extensors in terminal phalanges.

strain than that with which the patient was infected. In the second case the strain of the hemolytic streptococcus of the recipient was undetermined. From my experience with immune transfusion the benefit derived from the donor's blood may not be solely attributable to the specificity of the strain of the hemolytic streptococcus. It would seem that the whole blood of the donor may at times contribute actively phagocytic leucocytes and an opsonin which does not demand absolute specificity of strain. There may be other factors which con-

tribute to the efficacy of such nonspecific transfusions.*

The first of the two streptococcus cases illustrated in the film showed the right hand of a young physician. I saw him twelve hours after the middle finger of the right hand had been widely opened. The finger was then completely gangrenous. The white count had fallen from 20,000 to 9,000 and he was desperately ill. The finger was disarticulated, the proximal portion of the flexor sheath opened and the flaps left wide open for dakinization. Two immune transfusions were performed using as donors individuals who had recovered from hemolytic streptococcus throat infections. I am indebted to Dr. Lyons, surgical resident at the Massachusetts General Hospital, for bacteriological studies in this case. The strain of hemolytic streptococcus was not the same in either of the donors or the patient. The patient's leucocytes showed practically no phagocytic activity. However, after the second transfusion the patient improved rapidly. Active motions of the thumb and remaining fingers were started ten days after the disarticulation. The film was taken nineteen days after disarticulation. Belt-knotting for finger exercise was started fourteen days after disarticulation. He resumed practice five weeks later. Subsequently a plastic will be done on the stump.

The second of the two streptococcus cases illustrated in the film shows the right hand of a butcher of fifty. He had sustained a punctured wound of the thumb two days previously. July 23, 1935, I saw him and performed an immediate drainage of the tendon sheath of the long flexor of the thumb, the radial bursa, and the wrist space. There was no evidence of involvement of the ulnar bursa. Culture showed hemolytic streptococcus. July 30 (one week later), the ulnar bursa became involved. The infection spread with such rapidity that within a few hours the little finger was gangrenous and the patient's general condition was alarming. The ulnar bursa was drained and an immune transfusion was given. The previous patient served as donor. Their bloods were compatible (Group II) but the strain of hemolytic streptococcus in the present case was unknown. However, improvement was immediate and the patient continued to make a satisfactory recovery. Active motion of the digits was started August 3 (ten days after the first operation and four days after the second operation). August 12 (thirteen days after the second operation) a film was made to show the incisions, motions, and the preservation of flexor tendons of the thumb and little finger. August 13 (the next day) the gangrenous little finger was disarticulated at the metacarpophalangeal joint and the flaps were left wide open for continued dakinization. August 31, discharged from hospital. September 7 (three weeks later) film was taken to show the condition of wounds, the degree of active motion, and the preservation of the flexor pollicis longus tendon. Subsequent progress was satisfactory.

In cases of infection of the finger sheaths and the radial and ulnar bursae the preservation of the involved tendons cannot be assured. If the mesotenon does not undergo necrosis or if its vessels do not become thrombosed, the tendon will survive. The indications, therefore, are prompt adequate incision, and drainage in such a manner that the tendon blood-supply is not jeopardized in sheath infections. I never dislodge the tendon from its bed. I prefer to use multiple small gauze drains impregnated with boric ointment, laid lightly into the wound to hold the mouth of the wound open, *not* pushed down beside the tendon.

*NOTE: My first immune transfusion was done in 1930. On April 14 I had operated upon a doctor desperately ill with a hemolytic streptococcus infection of his left index finger sustained by a needle prick. On June 11 I performed an appendectomy with drainage on a child. The culture proved to be a hemolytic streptococcus. Some days later the child went into collapse. I transfused the child with the doctor's blood. The immediate benefit was not less than spectacular. The strain of hemolytic streptococcus was unknown in either donor or recipient. I fully realize that this procedure was not scientific, but the happy result in this case has been happily repeated in other cases since then

DISCUSSION

DR. ALLEN G. RICE, Springfield, Mass.: *Mr. President and Gentlemen*—I think it has been eight years since we have listened to anything about the hand. It seems to me a rather important aspect of our work, especially in these days of industrial accidents.

After this very beautiful demonstration, I hate to say anything that would blur this excellent picture which Dr. Harmer has left us. All I feel confident to say is that I think there is a place for splints at times. I do not think you can make any hard and fast rule that splints are to be removed forty-eight hours or three days or five days later. I think it all depends on the case. It is pretty hard sometimes to judge just when the splints should be removed and when to start motion. I think perhaps the best criterion is the patient himself.

If the motion is slight, he will have very little pain. I am inclined to leave it to the patient himself, to make him start motion. If it hurts him too much, stop. Then begin again and let him move up to the point when it causes him pain.

Otherwise, I think I have very little to say, except to agree heartily with Dr. Harmer's points. He spent most of his time on tendon sutures. Some of us out in the crossroads see a lot of bad hand injuries. It is surprising to see the results that can be obtained if you let them alone. The tendency sometimes is to do some sort of surgery on them.

The very tip of the finger can, I think, best be treated with a celluloid cuff that projects beyond the end of the finger. Let it fill up with blood and leave it alone. That finger will mold itself a pretty satisfactory pulp.

Another instance where the pulp has been entirely cut away, I had a little while ago a young lady who played the violin. She was slicing bread and sliced the whole pulp off the end of her left finger. That left finger of hers was rather important.

In casting around for a place that had no hair and would look pretty well, I bethought me of the toe. I took the pulp off the toe and put it on the end of the finger, and, much to my surprise, it worked pretty well. Fingers that are cut off by saws with nothing left except perhaps one little artery on the side, can yet be saved. Wrists with not only tendons gone, but both arteries, can be saved. There is a little bit of an artery that runs down the middle. That is sufficient, lots of times, to preserve that hand.

I have enjoyed this paper very much.

PRESIDENT JOHNSON: Is there any further discussion?

DR. JOHN HOMANS, Boston, Mass.: I am not quite sure that it is germane to this discussion, but it occurs to me to speak about a method of filling a considerable gap in a tendon which I have used in the case of one of the extensors of the thumb. It is perhaps a little clumsy but permits very early use. If a piece of fascia lata is taken, fairly narrow and split into two tails at either end, those two tails

can be wound around in a spiral manner, one against the other, so as to require very little sewing. Such an attachment can be made to each stump of the divided tendon with very few stitches, and the harder the pull, the tighter the lacing. So one feels very comfortable about making early use of such a union.

PRESIDENT JOHNSON: Is there any further discussion? If not will you close the discussion, Dr. Harmer?

DR. HARMER: The title of my paper suggested the boundaries of some of the early grants of land in this country, bounded on the east by the Atlantic Ocean and the west by the setting sun. I have tried to pick out just a few points to illustrate active motion or active motion with partial restriction of antagonistic tendons.

What Dr. Rice said is the same criterion which I use. If the flexor tendons are severed, we will say, at the wrist with nerve injury and coincident repair has been done, I have the patient move the fingers in flexion up to the point of pain, as Dr. Rice said, but prevent the fingers from being carried into extreme extension.

As for the practice of conservatism in hand injuries, that cannot be too strongly emphasized. Hands which look desperate from crushes should have simple débridement done, followed by dakinization even in the presence of fractures. Treatment of the soft parts may be more important than treatment of the bones. The fractures, I think, at first should be disregarded and no primary effort made to align them. What we are more concerned with is the ultimate function of the hand rather than its appearance. Later work can be done to improve the appearance.

Dr. Homans' remark about the tendon graft is interesting. I have used fascia lata where I have had a good many tendons to repair. In short gaps I like to use tendon for the grafts rather than fascia lata, although the fascia lata lace, as was said, gives you a feeling of security that it is not going to pull out.

I have been in the habit of using the palmaris longus, when it was present, or strips from the side of the tendo achillis or the plantaris, when present.

THE ACTIVITY OF THE URINARY BLADDER AS MEASURED BY A NEW AND INEXPENSIVE CYSTOMETER*

BY DONALD MUNRO, M.D.†

IN an attempt to arrive at a better method of treating urinary bladders paralyzed as the result of spinal cord injuries, it was soon demonstrated that cystometrograms were essential. A Rose cystometer[1] was tried but proved unsatisfactory for various reasons. In the first place the small graph did not show the individual contractions in sufficient detail. Secondly, the rapid fill inherent in this instrument masked the true contractile activity of the detrusor muscle and set up false levels of intravesical pressure. Finally these defects plus the limited amount of funds at my disposal made its cost prohibitive. At that time there was no other cystometric apparatus available except the experimental one devised by Denny-Brown and Robertson[2]. I therefore adapted the tidal drainage apparatus which I had described before the New England Surgical Society in 1934[3] for this purpose. I found that this provided a sufficiently accurate though crude home-made instrument. It was simple to operate and permitted the taking of observations at any desired rate of fill. Graphs made with its help were detailed enough to demonstrate minor as well as major variations in the contractile wave and it was constructed with the expenditure of about two hours of labor and $5.00 worth of material. It was used at first in the study and treatment of abnormal bladder activity associated with spinal cord injuries. A preliminary report of these findings was made at the time the tidal drainage apparatus was described as noted above. Since then this cystometer has been more widely used in a variety of normal and diseased conditions and in addition as a major therapeutic aid in the treatment of an increasing number of spinal cord injuries. This paper, however, is limited to a description of the cystometer and the presentation of the data derived from the study of the activity of thirty-one normal bladders. The observations were made on men, women and children and by a variety of operators.

With the antecedent tidal drainage apparatus, infection of the genito-urinary tract and the associated nursing problem of the constantly wet bed in spinal cord injuries of all types have been completely eliminated during the past two and a half years. With this additional cystometric modification and an associated graphic background of normal human urinary bladder activity, the atrophic overdistended, and the hypertrophic shrunken bladders have also been eliminated. As a corollary, information is now available regarding the tonic response of the bladder muscle at the start of filling, the curve of increase of intravesical pressure with continuing distention, and the frequency and type of reflex motor response initiated by a standard rate and amount of fill. By inference and because of the work of Denny-Brown and Robertson[2], the reflex activity of the two sphincters and the voluntary control over the external sphincter are also demonstrable. This should make obsolete any such inaccurate classification as is implied by the term "neurogenic" and lead to a more intelligent understanding of the requisite treatment in certain types of bladder disease.

*From the Neurosurgical Service, Boston City Hospital.
Read in abstract before a joint meeting of the Philadelphia Academy of Surgery and Boston Surgical Society at Boston, Massachusetts, February 3, 1936.

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*The Cystometer.** (Figure 1.) In its essentials the cystometer consists of a large U tube with a side opening in the proximal arm. The latter is connected with the bladder by a length of tubing attached at its distal end to an inlying catheter. To the upper end of the former, and 70 cm. above this side outlet, an air-tight "Murphy dropper" is attached to a 500 cc. container graduated in 25 cc. amounts. Above the

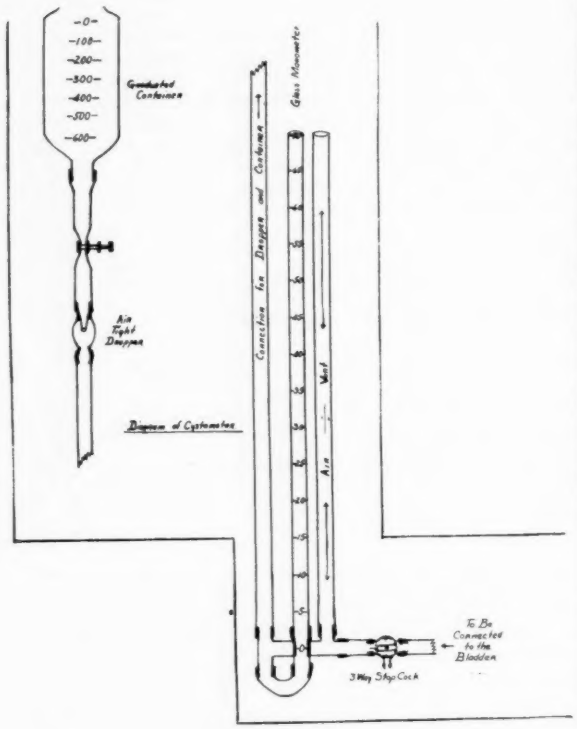

FIGURE 1.

dropper an adjustable compressor surrounds the connecting tube. This regulates the rate of flow from the container to the dropper and permits its estimation in terms of drops per minute. The optimum rate has proved to be about 120 drops per minute. The other distal arm of the U tube is formed by a 70 cm. glass tubing of 4 mm. bore marked every ½ cm. from a zero point which is set close to the level of the bladder connecting arm. Introduced into the bladder connection is an air vent which is of the same length as the manometer together with a three-way stopcock which allows fractional emptying of the bladder.

With the patient as nearly as possible horizontal, readings are taken with the apparatus hung on a stand at such a height that the zero point on the manometer is at the level of the patient's pubis. Under aseptic precautions the patient is catheterized, the bladder emptied and the catheter fixed in the urethra with the eye just inside the internal sphincter. In males it is preferable to use a soft rubber rectal tube of appropriate size because of the terminal opening. With the catheter attached and clamped off close to the meatus the cystometer is filled to the zero point with irrigating fluid from the graduated container. After making sure that the air is all out of the system the catheter is unclamped and the test filling started by adjusting the dropper so that the flow is at the rate of 120 drops per minute. Inasmuch as the system is air-tight and the bladder and manometer contents are in hydrostatic equilibrium, changes in the intravesical pressure will be registered on the manometer as rises and falls of the column of fluid contained therein. Contraction of the bladder wall or increase in this pressure will cause a rise in manometric fluid and relaxation the opposite. Respiratory excursions are visible constantly as are sudden alterations in intra-abdominal pressure when they occur. By starting the observations with the cystometer filled and the level of fluid in the container at 0 it is easily possible to make readings of intravesical pressure on the manometer scale simultaneously with notations of the amount of fluid that has emptied from the container into the bladder. These figures are recorded in parallel columns at the time of the observation. Later, they can, if necessary, be transferred to coördinate paper in such a way as to produce a graph which has for its abscissa or horizontal arm the fluid content of the bladder, and for its ordinate or vertical arm the intravesical pressure at a corresponding moment. This apparatus is admittedly much less accurate than that of Denny-Brown and Robertson but on the other hand is considerably more accurate than the Rose cystometer. Furthermore its accuracy is well beyond the requirements of daily practicality. Although there is a positive inflow pressure, the slow rate and the additional safety valve effect as provided by the open ends of the manometer and air vent prevent significant artificial alteration of the measurements. The readings of pressure are isometric below 70 cm. or in accordance with the height of the air vent. In the presence of a leak about the catheter or overflow from the manometer or air vent above 70 cm. they become isotonic. When recording, each change in intravesical pressure, as evidenced by a rise or fall in the column of fluid in the manometer, is noted. The corresponding increase in bladder content as evidenced by a fall in the level of fluid in the graduated container is also listed. Care must be exercised to make the recordings in such a way as to differentiate the step rises of pressure as seen in the normal bladder and the steady rise and fall to and from a peak as seen with uncoöperative patients or in completely atonic bladders.

After completing the observations detailed

*A commercial form of this cystometer has been constructed by and is obtainable from the Randall Faichney Co., Inc., 123 Heath Street, Roxbury, Massachusetts.

above, if the stopcock in the bladder connection is set at any predetermined level *above* the zero point, the full bladder may then be made to empty against positive pressure. With the stopcock *at* zero and no further emptying a true picture of the residual urine in the given case may be obtained by then withdrawing by siphonage (with the stopcock *below* zero) and measuring any remaining bladder contents.

Observations. Measurements as outlined above have been made with this apparatus on thirty-one patients who had at the time no bladder abnormalities whatsoever. Twenty-four of these thirty-one had never had before, nor did they have after the observation, any subjective or objective vesical abnormality. Of the remaining seven, three were made following presacral neurectomies for dysmenorrhea (through the courtesy of Dr. Frank Pemberton), three after cervical hematomyelia with various degrees of fracture dislocation of the cervical spine and one, three months after a compression fracture of the second lumbar vertebra.

In general it was possible to demonstrate three types of normal bladder activity as expressed in graphic form. The first may be considered as the true expression of the pure normal segmental reflex activity of the bladder in that the contractions were not inhibited by psychological influences. (Figure 2.) There were ten of these observations of which one is illustrated. These were made on patients who, because of associated brain tumors (4), senility (1) or low intellectual level (5), were characterized by an absence of the psychological inhibitions commonly seen in the social relationships of normal individuals. They were conscious, clean in their personal habits, completely cognizant of the need for a bedpan or urinal but their judgment in relation to such problems was conspicuous by its absence.

The graph in this type of response shows

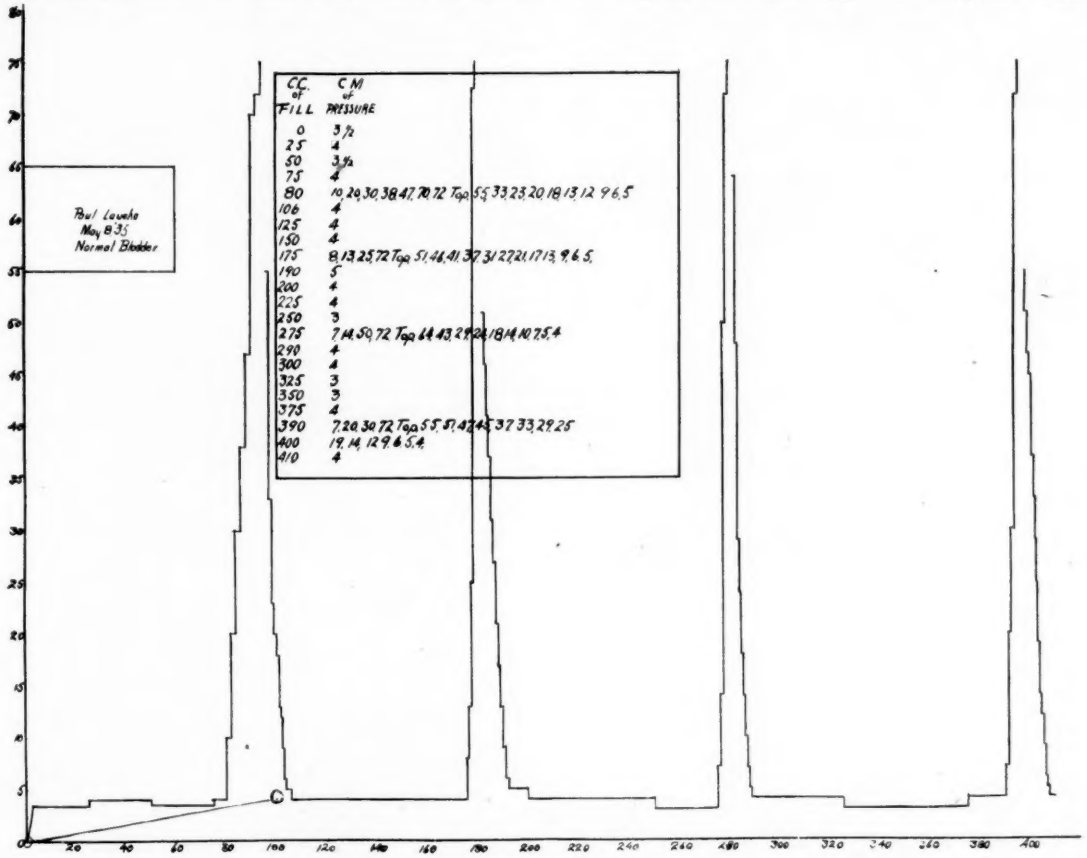

FIGURE 2.

that a fairly constant amount of stretch of the vesical wall as measured by cubic contents of the bladder produces a sudden sharp rise in intravesical pressure. This either ascends at once to an emptying peak or reaches there by the imposition of several increasingly stronger contraction waves one on the other. After a sufficient leakage has taken place the intravesical pressure descends again by steps to a slightly higher base than previously if the leak has been small, or to the original basic level if the leak has been larger. These peaks of contraction are uniphasic with a plateau at the apices and are more or less evenly spaced. The intervals between are given over to a succession of periods of distention against a constant resistance, interrupted at intervals by waves of contraction which do not produce emptying but only raise slightly the level of resistance to

further distention. Because of the variations due to leakage it was impossible to include these figures in the average graphs made up from the other two series. Similar graphs (not reproduced here) have been obtained in cases with proved transection of the thoracic cord. Here there was necessarily an associated anatomical severance of the bladder from the influence of higher intellectual centres although detailed studies showed normal function and anatomy of all parts of the genito-urinary tracts. These graphs also correspond within

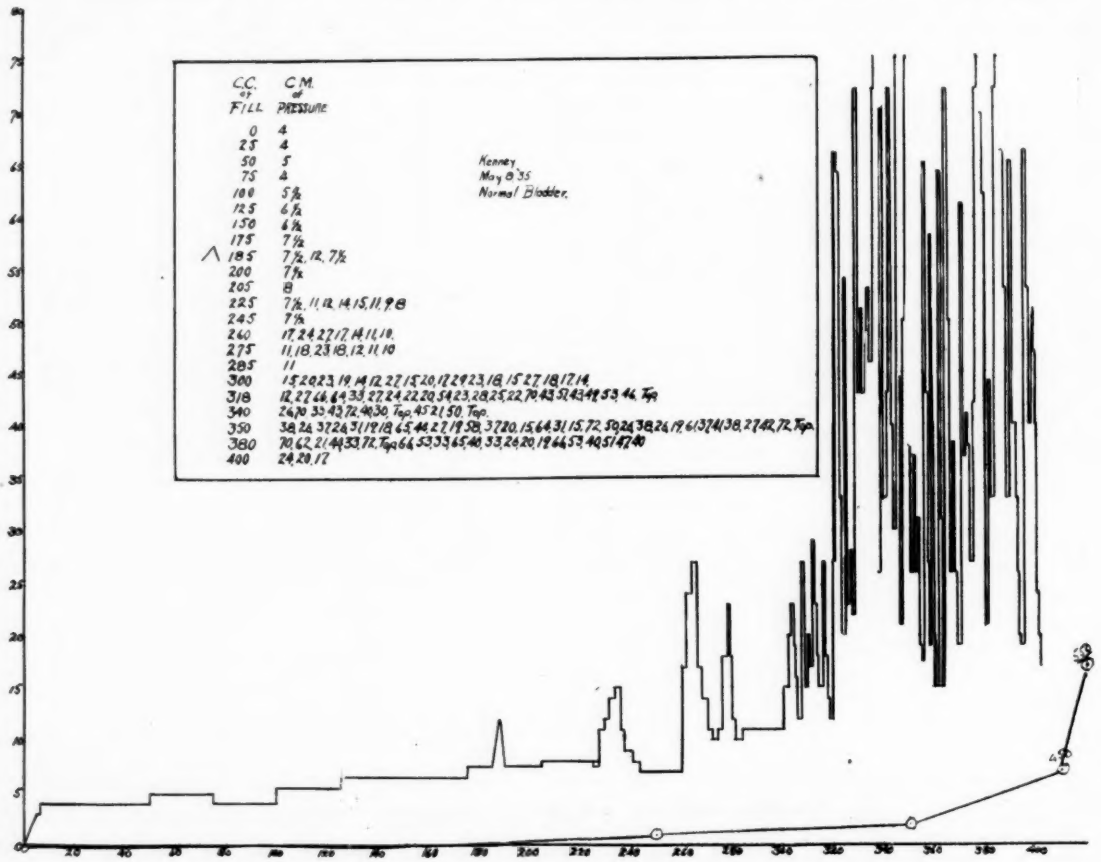

FIGURE 3.

the limits of comparison of the two methods, to the observations of Denny-Brown and Robertson.

The second type of normal bladder activity is that in which inhibitions from higher levels act to prevent detrusor contractions until the tetanic level of stretch of the muscle has been reached. (Figure 3.) With slow filling and without mechanical obstruction of the outlet every bladder will undoubtedly sooner or later reach this level under these conditions. However, because in these observations the amount of fill of the bladder at any one test has been arbitrarily set at 400 cc. or less, it has been possible to separate this group as those bladders which go into tetany within the limits of that fill. Here the intravesical pressure slowly rises. This is associated with stretching of the detrusor muscle interspersed with contraction waves only large enough to increase the resistance to further distention until at a certain critical point any given wave may suddenly build itself up to emptying levels. Instead of subsiding to the former precontraction pressure level after partial emptying, other large waves immediately follow in rapid succession. This gives the appearance of tetany on the graph and in the manometer, a condition that in all probability is actually present. In my experience, this type of graph has not been duplicated outside of a normal bladder and conforms as well to certain of the data obtained by Denny-Brown and Robertson. There were eight such patients. All had normal bladders, the graphs of which are represented by the one reproduced herewith. (Figure 3.) (Figure 5A.)

The third type of normal bladder activity is that in which emptying contractions are totally inhibited until after 400 cc. or more has been added to the empty bladder. (Figure 4.) There were eleven of these without previous bladder abnormality with an additional two in whom the graph was the same although made after pre-sacral neurectomy. These base lines duplicate those of the two other types of graph (figure 5B) and also conform to certain of the

data elicited by Denny-Brown and Robertson. From the combined data of these last two groups, a high, low and average base line for normal intravesical pressure has been determined. (Figure 5C.) Attention should be particularly directed to the shape of the curve and more especially to its rise through a series of steps. The degree of tonus set up in response to a minimum fill is also both characteristic and important. These factors together with uniphasic, more or less regularly spaced or tetanic contractions with plateau tops occurring in the absence of postural or psychologic inhibitions characterize the normal human detrusor muscular activity.

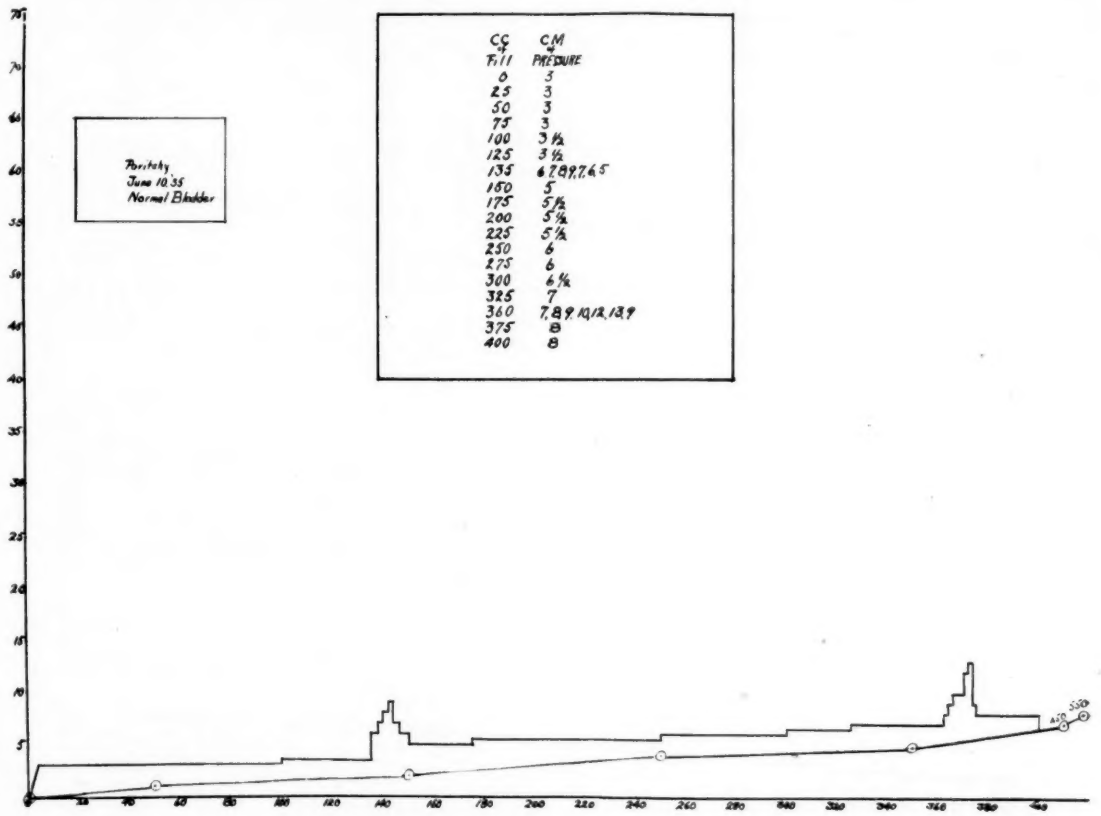

FIGURE 4.

Artificial Variations. There are two common artifacts that, unless recognized, may confuse the interpretation of the graphs. The first has to do with the rate at which the bladder is filled during the test. (Figure 6A.) This may produce an artificially high level for the base line and will completely obscure the normal step appearance that goes with increasing fill. Inasmuch as these variations may both be symptoms of interference with the normal vesical neuromuscular reflex, particularly in certain spinal cord diseases or injuries, it is important to identify the cause of their appearance accurately. The second is due to lack of coöperation on the part of the patient, especially if it takes the form of physical activity. This will produce sudden rises and falls of the fluid level in the manometer (figure 6B), which if observed carelessly may be interpreted as detrusor contractions. More careful study will demonstrate, however, that they lack the plateau at the apex which is an essential part of a true contraction and also do not show any steppage in either the rise or fall. They are due to sudden contractions of the muscles of the anterior abdominal wall and are the graphic evidence of an increase in intra-abdominal pressure transmitted directly through the bladder and its contents.

It is not appropriate at this time to consider the various other graphs that are associated with bladder pathology. However, for purposes of record, it can be stated that with this apparatus it has been possible to differentiate acute cystitis, a contracted hypertonic bladder, a non-contracted hypertonic bladder, a completely atonic bladder, destructive disease such as carcinoma of the wall of the bladder and finally the different stages of resumption of bladder function after spinal cord injuries.

Discussion: A detailed review of the studies which have led to our present understanding of the physiology of micturition cannot

be undertaken in this place. Suffice it to say that available knowledge is much greater than is commonly made use of. Experimental and other work done by such men as Learmonth[4], Holmes[5], Quinby[6], Graves and Davidoff[7], Graves[8], Langworthy et al[9, 10] and many others have cleared up many of the broader problems. Finally Denny-Brown and Robertson[2] with an ingenious and extraordinarily accurate apparatus have, with the help of normal human subjects, dealt with and settled many of the hitherto misunderstood details. The result of all this work may be summed up as follows: Micturition takes place because of the reflex contraction of the detrusor muscle accompanied simultaneously by a reciprocal relaxation of the internal urethral sphincter and followed by a further reflex relaxation of the external sphincter. This succession of events is started by a sensory impulse derived from a given amount of stretch of the bladder wall. This impulse reaches the sacral segments of the spinal cord by way of parasympathetic or pelvic nerves, is shunted in the cord to the motor neurone of this reflex arc and reaches the detrusor and internal sphincter musculature simultaneously as a motor impulse. As noted above, this produces contraction of the former and relaxation of the latter. This relaxation of the internal sphincter serves in its turn as a sensory impulse which again travels to the sacral segments of the spinal cord to leave as a motor impulse by way of the efferent side of a second reflex arc which this time includes the pudic nerves. This final impulse relaxes the external urethral sphincter. This activity is all mediated through normal neurological connections that lie wholly in the lower segmental levels of the central nervous system. It provides for the emptying of a given bladder in response to the purely automatic impulse elicited by a given amount of stretch of that particular bladder wall. If this constituted the whole story, however, the vesical storage facilities would be limited below known normal levels. This greater storage capacity is provided through the control of what would otherwise be emptying contractions of the bladder wall, by the impact of inhibitory impulses originated at suprasegmental levels. These impulses find their origin in unusual or inconvenient psychological or postural circumstances. They pass down the cord to act probably on the afferent part of the parasympathetic reflex arc. As a result, relatively great degrees of vesical distention may be reached before the urgency of the reflex detrusor contraction results in emptying. This storage capacity may be still fur-

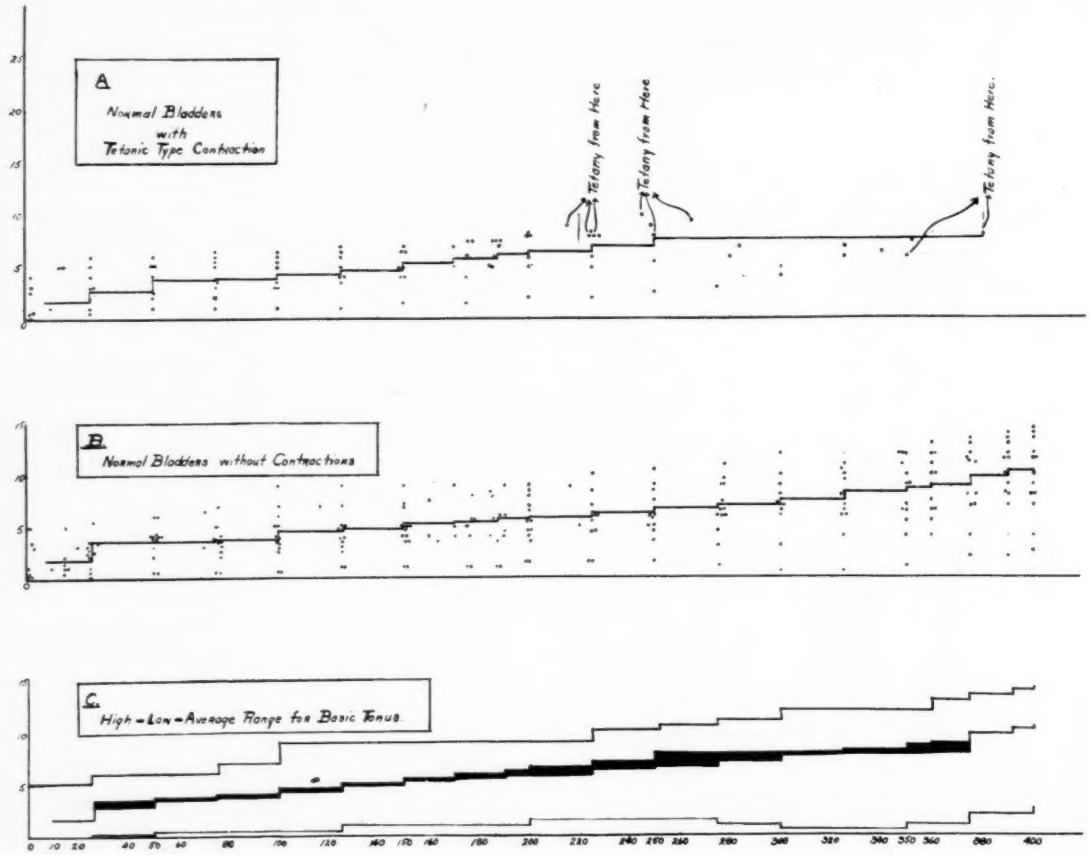

FIGURE 5.

ther increased by the voluntary contraction of the external sphincter. This can be imposed at will on the reflex relaxation that would otherwise take place. As a corollary to this it becomes apparent that facilitation of micturition is attained only by the control of the inhibitory impulses from the higher central nervous system levels. Only in this way can the underlying reflex activity leading to emptying resume its normal rhythm. It is furthermore true that relaxation of the external sphincter takes place only reflexly. The functions served by the cause my observations were made with a method of fill that approximated the normal. They were both isotonic and isometric in type. On the other hand direct observations were made of the detrusor contractions only and do not include any sphincteric data that is other than inferential. They demonstrate again that the bladder distends by periods of stretch which are brought to a close by the imposition of contraction waves. This stretch occurs in the presence of a fundamental basic tonus, while the contractions raise the level of this fundamental

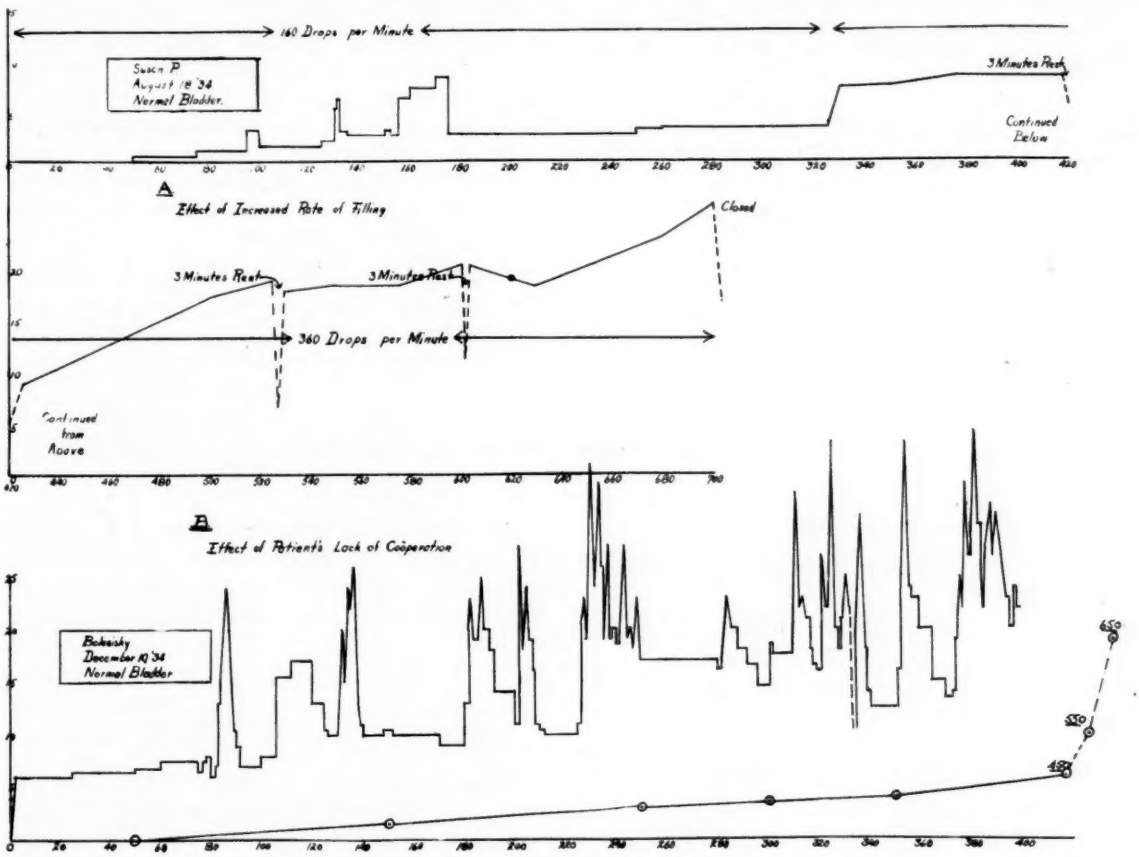

FIGURE 6.

sympathetic connections are still in dispute with no final evidence available one way or another. Also in doubt is the presence of an autonomic plexus analogous to Auerbach's plexus and lying wholly within the limits of the vesical wall. What evidence there is, however, is to the effect that this structure, if present, functions only in the event of an otherwise complete denervation of the entire bladder and its sphincters.

The data outlined above and obtained as described from the study of thirty-one normal bladders strongly confirm this summary of the physiology of micturition. They also agree with that obtained with more accuracy though from fewer individuals by Denny-Brown and Robertson. This is the more significant be-

tonus by steps until such time as the critical degree of distention for the individual bladder is reached. Here further action depends upon two factors. If inhibitory psychological or postural impulses are removed because of disease or by division of the connections linking up the suprasegmental intellectual levels to the bladder, an emptying contraction will take place. This may take the form of a single large wave or that of a pyramid of successively greater waves. If on the other hand suprasegmental inhibitory impulses are allowed full play, what would otherwise be an emptying contraction is flattened out and produces no more effect than the antecedent non-emptying ones. The resulting distention will then continue until such time as the inhibition is removed by extravesical

cause or until, because of pain in or overdistention of the bladder, the motor response no longer remains under control. This is succeeded by a tetanic type of contraction which lasts until the excessive distention has been reduced to a level where the inhibitory factors can again assume control.

While no direct evidence of sphincteric action is available as the result of this work it can be assumed with safety that the similarity between these findings and those of Denny-Brown and Robertson as regards the detrusor action justifies the conclusion that here too detrusor contraction was accompanied by reciprocal sphincter relaxation. In addition it is certain that in all these cases voluntary control of the contraction of the external sphincter was easily demonstrable at the time of catheterization. Furthermore anal and glans reflexes, the pathways for which are known to lie in the pudic nerves, were also always active. This would seem to imply that the reflex which led to relaxation of the external sphincter following detrusor contraction was also normal and active inasmuch as the same anatomical connections with the spinal cord were involved.

These data give no information regarding the impulses that traverse the sympathetic system connections per se and offer neither confirmation nor denial of the presence of an autonomic intravesical plexus.

CONCLUSIONS

1. A simple inexpensive portable cystometer is described.

2. Simultaneous readings of approximate content of the bladder and intravesical pressure, when made with this cystometer, give sufficiently accurate information about the activity of the detrusor muscle and the internal urethral sphincter to identify any deviations from normal.

3. When properly charted on coördinate paper of a sufficient size these figures will provide a graph of this activity.

4. Normal involuntary reflex activity of the external urethral sphincter can be checked by the activity of the anal and glans reflexes. It parallels that of the internal sphincter.

5. The normal ability of the patient voluntarily to contract the external sphincter can be measured by his resistance to the passage of a urethral catheter.

6. Normal detrusor activity must approximate the following requirements:

(a) The initial tonus of the detrusor muscle set up in response to less than 10 cc. of content will vary from ½ to 5 cm. of water pressure.

(b) Distention of the bladder proceeds against a background of tonus which is constantly being increased by additional increments added by means of waves of contraction that vary from the lowest possible level to heights that will produce complete emptying.

(c) This basic level does not normally rise above 15 cm. of water with the first 400 cc. of fill.

(d) Emptying contractions are uniphasic with a plateau at their apices.

(e) While normal bladder activity is fundamentally a function of a pure spinal segmental reflex arc it can be inhibited up to the point of detrusor tetany by impulses from suprasegmental levels. These impulses are set up as the result of unfavorable postural or psychological influences, or by voluntary contraction of the external urethral sphincter.

7. Graphs of normal bladder activity made with this cystometer fall into three types: (a) one in which spaced emptying contractions occur throughout the experiment; (b) one in which emptying contractions occur toward the end of a fill of 400 cc. in the form of unspaced constant tetanic contractions and (c) one in which during a fill of 400 cc. there is a complete absence of emptying contractions.

8. Facilitation of micturition depends in part at least upon the repression of conscious and unconscious inhibitions. This permits the normal underlying reflex activity to resume its rhythm.

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NEW HAMPSHIRE MEDICAL SOCIETY

MANAGEMENT OF SKULL FRACTURES*

How Can the High Mortality Rate Be Reduced?

BY HARRY E. MOCK, M.D.†

IN a previous publication‡ the author has shown that at least 125,000 proved skull fractures occur annually throughout the United States. The mounting casualty rate from automobile accidents during the last five years has undoubtedly raised this number to more nearly 150,000 skull fractures per year. Wherever trains run, automobiles are driven, airplanes fly, horses are ridden or stairways are built, there you may have skull fractures. The victims of this trauma frequent our smallest, as well as our largest, hospitals. In fact, I have knowledge of certain small but well-equipped hospitals located near extremely busy through highways where the number of skull fractures treated exceeded the number in certain of our well-established neurosurgical centers. Thus, it is evident that skull fractures constitute a problem for the general physician and surgeon, as well as for the neurological surgeon. It was this fact, combined with the location of our hospital, permitting the development of a large traumatic service that caused this problem to become my chief surgical hobby. The gradual but steady reduction in the mortality rate which is being reported from a constantly increasing number of clinics, both from neurosurgeons and general surgeons, shows that the campaign for better management of skull fractures is succeeding.

The term "skull fractures" rather than "head injuries" is used purposely by the author but it must be emphasized that *be it cranial, cerebral, or craniocerebral,* the treatment of the injury is identical. The fact cannot be overemphasized that a given head injury, without any x-ray or other evidence of skull fracture, may require just as close observation, just as painstaking management, and even then may prove fatal.

However, no uniform statistical study of this problem can ever be made if "Head Injuries" is the basis of reporting cases. One man's judgment as to the seriousness of a head injury before it should be included as a "possible clinical skull fracture" differs materially from another's judgment. The mortality rate is the best measuring rod for gauging the results of treatment and whether advances are being made in treatment. Thus, if one author builds up his statistics on absolutely proved skull fractures, his mortality rate will be more favorable, because many head injuries which are practically positively proved skull fractures will die before absolute proof is obtained. Again, if an author includes a large number of head injury cases in his statistics because he feels that they are clinical skull fractures, his mortality rate will likewise be more favorable, because the death rate in several large series of head injury cases without fractures has proved to be considerably lower than when fractures existed. On the other hand, if an author includes in his statistical study not only his definitely proved skull fractures, but from 3 per cent to 10 per cent of cases dying before proof can be obtained but which are so positively clinical skull fractures that they must be thus classified, then it is evident that a truer, although higher, mortality rate will result.

In the years 1931 to 1934 inclusive, the author presented a Skull Fracture Exhibit in the Scientific Exhibits of the American Medical Association. In the first Exhibit at Philadelphia in 1931 he presented 100 consecutive cases of proved skull fractures from his own clinic at St. Luke's Hospital. At that meeting and at subsequent meetings many physicians and surgeons throughout the country, many of them from your New England States, consented to coöperate with him by reporting their consecutive skull fracture cases on a common blank furnished by the author. Likewise several hospitals reported their cases and six hospitals allowed the author and his two associates, Dr. Reed Morrow and Dr. Charles Shannon, to study their consecutive head injury files for a period of five years, in order to select the proved skull fracture cases. The facts and impressions presented in this article therefore have been gleaned from a study of 2595 cases of positively proved or practically positively proved skull fractures collected from the following sources:

1. Two hundred cases of skull fractures entered on the author's service at St. Luke's Hospital. Ninety-two per cent of all these were positively proved skull fractures. The remaining cases were so obviously skull fractures that they had to be included, although they raised the death rate 3 per cent.

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 8, 1935.

†Mock, Harry E.—Senior Attending Surgeon, St. Luke's Hospital. For record and address of author see "This Week's Issue," page 653.

‡Management of skull fractures and intracranial injuries. J. A. M. A. 97:1430 (Nov. 14) 1931.

2. Eight hundred and fifty cases collected from approximately 100 individual surgeons and twelve hospital staffs reporting on a uniform blank prepared and sent to them; approximately 1250 cases were submitted as skull fractures, but 400 of these were ruled out, as they did not fulfill our requirements for positively proved or practically positively proved skull fractures.
3. Thirteen hundred and fifty-four cases were obtained from a survey of the records of six hospitals covering a period of five years in each hospital. Approximately 5,000 records were studied, and from these were culled the 1354 cases of positively proved or practically positively proved skull fractures.
4. One hundred and ninety-one cases were submitted to us from the neurological service of a large hospital.

These cases fulfilled the requirements for proved skull fractures.

My address given before your Society last May was built around forty-two lantern slides which enabled me to demonstrate my subject more clearly. Space will not permit the reproduction of all this material, therefore I wish to devote this article to that portion of my address which dealt with the question—"*How Can the Mortality Rate from Skull Fractures be Reduced?*" In order to develop this subject it is necessary to reproduce just two of those lantern slides.

CHART A.

HOW CAN HIGH MORTALITY RATE BE REDUCED?

A—IN FIRST 24 HOURS

45% of Deaths Occur in 1st 24 Hours

1—Greater knowledge concerning early management by rank and file of profession.
  a—Laity education—prevention and 1st aid.

2—Treat shock first—everything else can wait.
  a—Don't suture lacerations at once.
  b—Don't reduce fractures at once.
  c—Exception—a severe life-threatening hemorrhage.
  d—Least possible moving of patient chief essential.

3—Every serious head injury is emergency case.
  a—Requires immediate attention of attending physician.
  b—Early skillful examination — blood and urine.

4—Don't overlook less obvious, often more serious, associated injury.
  a—Two cases ruptured spleen — operated — lived.

5—X-ray every head injury—but don't x-ray:
  a—In presence of shock.
  b—In presence of delirium or deep coma.

6—Immediate operation of Skull Fracture seldom indicated.
  a—30 immediate operations—28 deaths.
  b—15 subtemporal decompressions in 1st 7 hours—15 deaths.

7—Treat every head injury as serious until proved otherwise.

8—Morphine lulls the surgeon, as well as the patient, to sleep.

The following table shows the source of my figures for the above chart, as well as for chart B.

TABLE 1

Source	Number	Total Death Rate	Death Rate 1st 24 Hours	Death Rate 1st 7 Days
Mock's cases	200	18.2%	46%	38%
Collected cases	850	26%	44%	42%
Hospital Records	1354	40.5%	45.5%	41%

After all is said and done the answer to this question of, "How can the high mortality rate from skull fractures be reduced?" has been the chief concern of the Skull Fracture Exhibit, as well as the chief motive behind the efforts of all men working and teaching in this field of head injuries. Recently I came across an unprinted monograph by Dr. Nicholas Senn, written in longhand in 1896. In this article he stressed the fact that skull fracture was a widespread condition which must more often be treated by the general physician rather than by surgical specialists. He pointed out that the early management of these cases, before a special surgeon could arrive on the scene, often spelled life or death for the patient. In other literature of his day there was a note of inevitableness in the discussion of the mortality rate from head injuries. During the last decade the attitude toward this condition has been more hopeful and without question, many suggestions in management have been made which, if adopted and logically carried out, have been and will be life-saving measures.

In recent years, it seems that a note of pessimism has crept into some of the articles, and again the inevitableness of death in a certain percentage of skull fracture cases is being stressed. For example, Dr. Walter Dandy (*J. A. M. A.* **101**: 774 [Sept. 2] 1933) states: "Give nature a chance and 70 per cent of all patients with severe (head) injuries will recover spontaneously. If left alone, the remaining 30 per cent will die. From this group perhaps one

third (one tenth of the total injuries) can be saved by subtemporal decompression if well timed and properly performed. The remaining 20 per cent (of the total) must be regarded as beyond redemption by any rational means available."

If it were possible to pick out the inevitable deaths, or those that remain beyond redemption by any rational means, the situation would be ideal. It would then be simply a matter of concentrating upon the other 80 per cent. Time and again, in commenting upon one of our deaths from skull fractures, we have found ourselves saying: "Well, that was an inevitable death, nothing could have saved him." Time and again we have thusly predicted concerning a certain patient but have continued to fight and, to our surprise, the patient has recovered. It is simply impossible for any man so to classify all skull fracture injuries that the profession can select the cases of inevitable death and the cases that might be saved. This being true, our attitude must be hopeful, watchful, constantly searching for greater knowledge, especially in the management of the early cases, the time period in which the death rate is the highest.

There is great need here for education of the laity, especially first-aid and prevention methods. Without question, the states must develop uniform laws which will control the types of automobile drivers, standards of ability for the drivers, uniform traffic regulations, especially speed control, and compulsory insurance that will provide hospital and medical attention from the very minute an accident occurs. During A Century of Progress I gave an address on Skull Fractures for the College of Surgeons at one of their public meetings. I know from the testimony of two persons who heard the discourse that on two different occasions each prevented the dumping of an unconscious patient with a skull fracture into the rear seat of an automobile for the purpose of conduction to a hospital. They covered these patients up, kept them warm and insisted upon an ambulance for transportation. Talks of this nature should be given all over the country to lay audiences. Better and more complete ambulance service must be established everywhere. The profession must join with the laity in a fight, first, against accidents, and secondly, for better emergency and subsequent care when trauma occurs.

The second point in Chart A, viz., treat shock first—everything else can wait, is a commonsense but life-saving slogan where skull fractures are concerned. Not so often now as formerly, we have witnessed these skull fracture cases brought into the emergency room at the hospital unconscious and covered with blood which has oozed from the ugly scalp wound. The patient is cold and clammy and in coma or at other times is fighting in delirium. The interne or doctor in attendance shows a one-track mind when he pays attention to this laceration. Time and again we have witnessed such a patient in deep shock, with two or three people restraining him while the attending physician proceeds to shave his scalp and suture the laceration. After this is completed, the patient is moved to the hospital bed, occasionally to the x-ray laboratory first, yet most of us have learned not to x-ray these cases at once. It is of equal or greater importance not to delay the treatment of shock, while the laceration is sutured. Formerly we had in Chicago a hospital devoted solely to emergency treatment. None of the patients were kept in this hospital all night. I have seen several cases transferred from this hospital to St. Luke's while in shock and coma but with their scalp wound sutured. Two cases thus transferred developed a marked erysipelas while several cases developed scalp infections from the immediate suturing of the scalp wound. It is not only the best treatment to treat the shock first and suture the laceration later, but it is better treatment to suture the laceration after one has had a better chance to cleanse it and débride it and to make sure that a compound fracture does not exist.

The following case illustrates "b" under 2 in chart A., viz., the immediate reduction of fractures in the presence of skull fracture and shock.

Mr. B. was admitted to one of our best hospitals in coma and apparently with a head injury and a fractured femur. For the shock he was given a hypodermic of morphine at once. He was then taken from the emergency room to the x-ray laboratory where an x-ray of both the skull and the femur were made. Both showed fractures. The fragments of the femur showed little displacement and would require little manipulation and anyway the patient was in coma and would feel nothing. He was therefore taken to the nearby plaster room where a body and leg cast was applied. He was then moved to his hospital room. He was then in even greater shock, likewise he was extremely restless. Therefore another hypodermic of morphine was given. When this patient was seen by one of our best neurological surgeons one hour later, the patient had Cheyne-Stokes respiration, a rapid, thready pulse and died shortly after the specialist completed his examination.

Fractures can so readily be immobilized in a pillow splint or blanket splint or a Thomas splint, or even by a board splint that immediate manipulation and reduction or more elaborate splintage is never necessary or indicated when a skull fracture is suspected or when the patient is in shock, be it general shock from a fracture or cerebral shock from the head injury. Observance of this fact will save many lives.

Only occasionally is there a severe life threatening hemorrhage which requires the immediate attention of the surgeon, even in the presence of the shock. I have never observed a middle

meningeal or subdural hemorrhage that gave signs or symptoms of its presence to the extent that surgical intervention was necessary during the first few hours following the injury when the shock period is present.

The least possible moving of the patient is the chief essential in the presence of head injury, and especially when cerebral shock is present. In the author's 200 cases of skull fracture, shock in varying degrees was present in 90 per cent of the cases. I have seen it recently written that shock was not usually present in head injuries. This is not true, for in head injury sufficiently serious to cause the least disturbance of consciousness, shock is usually present. Remember, that it differs from the general picture of shock in that, added to this are the cerebral disturbances which cause respiratory depression, fluctuations in pulse rate and blood pressure, as well as the anoxemia peculiar to both general and cerebral shock. The following example best illustrates this point concerning moving the patient:

A young Jewish girl was admitted to the ward at St. Luke's Hospital in coma and with a definite head injury. Most of these serious head injury cases are first admitted to the ward. Many of them can afford and insist upon being moved to a private room. This move is never made with my consent until the shock has been combated and until the moving of the patient will not add insult to the injury. In this case, the parents insisted upon immediate removal to a private room. Upon my refusal to move her the family surgeon was called in on the case. He immediately ordered an x-ray which was made. When she returned from the x-ray room she was extremely restless and a hypodermic of morphine was given. Meanwhile, the family had arranged for a private room and the patient was at once moved to a distant part of the hospital to this private room. She died one hour later. This was not a case that could even be classified as an inevitable death. Unquestionably, the early moving of this patient was responsible for the disaster.

A physician who visited the Skull Fracture Exhibit in Philadelphia in 1931 and again visited the Exhibit in New Orleans in 1932, told me the following story: He practiced in a small village fifteen miles from the county seat where the hospital was located. One of the busiest through highways passed through his village where many accidents occurred. Prior to 1931 he was in the habit of rushing these accident cases, including his skull fracture cases, over to the county seat hospital. After listening to the first Skull Fracture Exhibit he changed his tactics. He stated that during the last year he had treated six head injury cases in his home, from a few hours to several days, before subjecting them to the long trip to the hospital. All six of these cases recovered whereas in the previous year all of his skull fracture cases died.

At the Skull Fracture Exhibit in Cleveland, at least five surgeons told me of treating skull fracture cases in nearby farmhouses for two days to three weeks before moving them into town to the hospital. One physician, discussing one of my early papers on Skull Fracture at Logansport, Ind. stated: "It will please the essayist to know that I have a skull fracture case out here in the country about eighteen miles. The accident happened ten days ago and I moved her into the nearest farmhouse. She is still alive and everybody is happy except the farmer. I won't move that girl until I know it is safe."

The above examples led to the development of my slogan—"A Live Skull Fracture in a Farmhouse is Better Than a Dead One in the Hospital."

Every serious head injury is an emergency case. No conscientious surgeon today neglects to respond immediately to a call from a patient, or from his interne, concerning a case of acute abdominal pain radiating toward the right lower quadrant. We have become thoroughly imbued with the emergency nature of acute appendicitis. It is just as essential to respond to the call concerning an acute head injury. Yet in reviewing the skull fracture cases from the records of six hospitals it was noteworthy that many a case admitted at night was not seen by the attending man until some time the next day. Several cases died before the attending surgeon's visits—one as long as twenty-eight hours after admission.

When should a head injury be considered serious and therefore an emergency case? Several conditions are here involved, chiefly the following:

(a) When the head injury, although apparently trivial, has occurred in a serious accident with potentialities of great damage.
(b) When there has been a loss of consciousness.
(c) When the case is sufficiently serious to be admitted to the hospital.

No matter what time of the day or night the call comes the physician or surgeon responsible for the case, or an experienced assistant if he has one, must immediately see that head injury case. He must make a careful, skillful examination without unduly disturbing the patient. He must look for associated injuries. The urine (catheterized specimen if impossible to obtain otherwise) and the blood must be examined. Why? Because occasionally the urine is bloody indicating the ruptured kidney or bladder; or the leucocyte count is high suggesting the possibility of an internal abdominal injury. The surgeon with his experience is better able to judge whether the trivial injury case needs to remain in the hospital. This decision should never be left to the interne. The surgeon should be the one to determine whether the case is able to be x-rayed at once. He should direct the treatment of the shock. He should remember that 45 per cent of his cases will die in the first

seven hours and therefore his close observation and direction of the treatment is needed most during this period. May the sleep of any of us be terribly disturbed if we fail to respond immediately to the call of the head injury case!

Many are the examples which I have collected, some in my own practice, illustrating the need of emphasizing these rules concerning the emergency nature of the head injury case:

(a) There was the case admitted to my service at three A.M. This patient, male, forty-five, was stuporous, but the interne could smell alcohol on his breath and felt he was drunk. The slight bump on the head seemed trivial. He was new on the service and we had failed to instruct him in the rule to call one of us on every head injury case (mistake 1). I was signed up to operate at eight A.M. and therefore did not visit this patient as soon as I reached the hospital (mistake 2). A blood count was made early that morning but of course I did not know that it was 26000 whites because of failure to visit the ward (mistake 3). At eleven A.M. the nurse phoned the operating room that the patient seemed to be dying. We hurried to the ward and found the patient in extreme shock, blood pressure 48, and pulse barely perceptible. Examination showed a rigid abdomen, dullness in left flank. The leucocyte count was now 36000. A diagnosis of ruptured spleen was made. In spite of the serious condition of the patient, I operated and removed a badly ruptured spleen. Preparations had been completed for a blood transfusion and this was done at once. A second transfusion was given at eight P.M. This patient made an excellent recovery. There were no signs of a skull fracture at any time other than the unconsciousness. However at the end of two weeks an x-ray of the skull showed an extensive linear skull fracture.

The failure to observe the "rules of the game" almost cost this patient's life.

(b) A serious automobile accident occurred. A husband, wife and their five-year-old boy were in the car. They were brought to a hospital and hurriedly examined. The boy was not hurt apparently. He sat on a chair in the waiting room while x-rays were made of the mother and father, both of whom had minor fractures, one of the clavicle and the other of the hand. No one paid further attention to the boy. The father and mother had been treated and were about to depart when the boy suddenly fell off the chair unconscious. He soon regained consciousness but could not talk and soon developed a paresis in the right arm and later in the leg. I was called, diagnosed a middle meningeal hemorrhage, operated, and found the same. The boy recovered.

This case shows how easily the true condition in the boy's case was overlooked and the potentialities of a catastrophe which existed if he had been allowed to depart.

(c) One of my senior medical students was externing at a small hospital at night. A boy was brought in about eight P.M. having been struck by an automobile and knocked completely across the street. He had a scalp wound but otherwise seemed unhurt. The surgeon who ran this hospital examined the boy and then told the externe to suture the scalp and let the father take his boy home. It was a "hit and run" accident, the family was very poor, and no one could be financially responsible for the case. The externe's suggestion regarding keeping the boy in the hospital under observation was therefore rejected with the excuse that "these kids are tough and he isn't hurt." About three A.M. the father came to the hospital stating that his boy was acting and breathing abnormally. The externe went to the home, found the boy stuporous, slowed pulse, and with stertorous breathing. He helped the father carry him to the hospital and the surgeon was called. Because of lack of financial means a police ambulance was called and the boy was transferred to the county hospital. He died a few hours after admission from an obvious middle meningeal hemorrhage and without the benefit of surgical intervention.

Mistake 1: Failure to keep under observation the apparently trivial injury from a serious accident.

Mistake 2: Undue transportation of the case.

Mistake 3: The apathy of the public and of the profession in not long since demanding a commonsense law which would compel all county commissioners to pay these small, poorly endowed hospitals to keep such patients as this boy, and similar emergency cases, until it was safe to transfer them to the county hospital. They could at least pay a rate commensurate to the cost of caring for the case in the county hospital.

(d) There is the case of the airplane pilot who crashed with four passengers. He helped get the injured from the wreck and helped to take them to the hospital where all were admitted, treated, and recovered. He only had a scalp wound which was sutured and he was allowed to depart. That night he became unconscious in his hotel room, was not discovered until morning, and died before they could get him to the hospital.

At least twenty examples have been collected of this type of head injury—scalp wound sutured, allowed to leave, later the cerebral damage manifested itself and death before anything could be done, or death in spite of the efforts of delayed treatment.

If the reader is tired of examples remember that the author is endeavoring to emphasize *how the high mortality rate from skull fractures can be reduced.*

The skull fracture is often so obvious that it completely explains the extreme shock and deep coma that are present and persist until the patient dies. Even the coroner feels that the cause of death is quite obviously skull fracture and therefore refuses to do an autopsy. However in these days of terrific traffic accidents, with

their multiple injuries, one must never be satisfied with a diagnosis of skull fracture alone until by careful and repeated examinations he has ruled out the less obvious, but often fatal, injury. Many a case of so-called inevitable skull fracture death might have been saved if search for the less obvious injury had been made.

The author has had two cases of ruptured spleen occurring with proved skull fractures in which splenectomies were performed with recovery. Dr. S. W. McArthur, at St. Luke's Hospital, diagnosed a ruptured liver in a young lady four hours after admission with a skull fracture and with the patient in deep coma. He operated, found a badly lacerated liver, packed the wound, and this patient recovered.

A surgeon told me at the Cleveland convention of a skull fracture case which he treated for twenty-one days; the patient recovered and was allowed to go to his home in a small town twenty-eight miles away. A week later this patient suddenly became very ill and died within a few hours. The surgeon went to this home, performed an autopsy, and found a ruptured spleen as the cause of death. Delayed hemorrhage from a ruptured spleen is not uncommon.

There is no need to devote much space to emphasizing point 5 in chart A. I am convinced that in the past too many cases were sacrificed by the immediate rushing of the head injury case to the x-ray. No skull fracture, and for that matter no other fracture, when the patient is in shock, should ever be x-rayed. Later, and as soon as safe, the x-ray should always be made, for it will prevent too early movement of an otherwise symptom-free patient; it will tell whether a depressed fracture is present and of a sufficient degree to warrant operative intervention; and it will show the location of the fracture, often a guide in both prognosis and treatment.

There are certain definite indications for operation in skull fractures as will be shown later. There are many borderline cases where it is extremely difficult to determine whether these indications are present. There are cases that are certainly nearing death where one feels that something surely can or must be done. The family is begging for action which fact often influences judgment. But from my own experience and from a review of the 850 cases reported to me and from a study of the 1354 hospital records one feels that this axiom holds true: *the immediate operation of a skull fracture, or the operation within a few hours, is seldom indicated.*

In my own experience there were three early cases operated, one, five, and seven hours after injury, who were robbed of any chance they may have had for recovery.

From a study of the reports of cases sent me by doctors from all over the country there were records of nineteen subtemporal decompressions, done within the first seven hours, with 100 per cent mortality.

From the hospital records, in spite of an exceedingly low operative rate, thirty cases of immediate operations were collected with twenty-eight deaths.

Dandy, who seems to favor operative treatment as the one definite thing that can be done in approximately 10 per cent of the cases, states emphatically that the longer the operation can be postponed the better the results.

Even in epidural or subdural hemorrhages the signs and symptoms of such conditions rarely develop within the first few hours. The author has had one case of middle meningeal hemorrhage that was definitely diagnosed and operated successfully six hours after the injury whereas in nine other cases, similarly diagnosed and successfully operated, the signs of this condition developed from two to thirteen days after the injury. As has been pointed out by Munro the signs and symptoms of subdural hematoma develop late as a rule.

Treat every head injury as serious until proved otherwise. This rule has been explained for the case which seems trivial and turns out seriously. On the other hand the rule does not mean that every head injury case must have a lumbar puncture or must be kept in the hospital for three weeks. In fact many of them are never hospitalized. But one should be quite positive, as a result of a careful neurological examination, that the patient is free of dangerous developments before deciding against hospitalization.

The majority of these questionable cases will turn out to be simple cases of concussion or possibly contusion—a differentiation often impossible to make. It is my habit to keep the less serious concussion case hospitalized and under observation at complete rest for one week, while the more serious, possibly contusion case is treated for ten days at least.

Morphine lulls the surgeon, as well as the patient, to sleep. This is just as true in skull fractures as in appendicitis where most of us have learned to avoid this drug. In addition morphine is a respiratory depressant and this effect added to the respiratory depression so often present in head injuries is sufficient to be disastrous. A survey among a great many brain surgeons has developed the fact that practically none of them ever use morphine either in skull fractures or other cerebral conditions. Experience has taught them its dangers. General surgeons should be governed accordingly.

It is noteworthy that in the author's cases as well as in a study of the entire remaining 2395 cases 40 per cent of the deaths occurred

in the first seven days. Concentrating his every effort to reduce the mortality rate during this period, one gains impressions concerning therapeutic effects which, with the accumulation of data, gradually merge into facts. Proof of the merit in these facts comes with the gradual lowering of one's mortality rate. Furthermore, it is noteworthy that every man who has concentrated upon the management of the skull fracture case has witnessed this same lowering in his mortality rate. This is true in the case

CHART B.

HOW CAN HIGH MORTALITY RATE BE REDUCED?

B—AFTER 24 HOURS

40% of Deaths Occur from 2nd to 7th Day

1—Greater knowledge concerning general management.
- a—Dehydration started early (after shock) and persisted in will reduce fatalities.
- b—Examine frequently—certain signs and symptoms, interpreted correctly, require life-saving special treatment.

2—The earlier lumbar puncture is performed, when indicated, the lower the death rate.
- a—Not indicated in presence of grave shock.
- b—Increased intracranial pressure—persisting after early dehydration—requires lumbar puncture.

3—Repeating lumbar punctures when operation is indicated increases fatalities.
- a—One case—coma; punctured; conscious; convulsions 3rd day; punctures repeated; death 4th day; autopsy subdural hemorrhage.
- b—Six cases—definite focal signs, 2 to 14 days; repeated lumbar punctures; all died; autopsies in 3—middle meningeal hemorrhages.

4—8% of cases require operation.
- a—Operative rate over 12% or below 6% increases mortality rate.
- b—The longer operation can be safely postponed the lower operative death rate.
- c—Subtemporal decompressions seldom indicated—in 942 cases, 27 such operations—22 deaths.

of the 150 physicians and surgeons who were sufficiently interested in the problem that they took the time to fill out the two-page skull fracture blank which we furnished them.

Temple Fay in his writings shows a reduction in his mortality rate from around 21 per cent to approximately 12 per cent in skull fractures and serious head injury cases.

George Swift in his writings and in personal communications shows a reduction in the mortality rate in the serious head injury cases cared for at Harbor View, Seattle, from around 35 per cent several years ago to 22 per cent five years ago and finally to 12.1 per cent for the last three years.

The author, having built his statistics on proved or practically proved skull fractures, naturally has a somewhat higher death rate than the above authors. He has no statistics upon his death rate prior to 1925. But since that time, concentrating upon the management of the skull fracture case, he published in 1931 a mortality rate of 20 per cent for proved skull fractures, counting every case that entered his service even though death occurred one minute later. Now his mortality rate is down to 18.2 per cent for proved skull fracture cases. Counting skull fracture cases and some 500 serious craniocerebral injuries in addition his mortality rate is down to 11 per cent.

The author's experience as well as the testimony of many others working on this problem is sufficient proof that the serious head injury case need not be left to nature; that there are several well-defined therapeutic measures which can be applied to the management of these cases and finally that the high mortality rate can be reduced if *greater knowledge concerning the general and special management of these cases is gained,* especially by the rank and file of our profession who are seeing most of the cases.

Comparison of the above mortality rates with table 2 will soon convince one that there is need for greater knowledge concerning these therapeutic measures. In the six hospitals whose records were studied the cases were cared for by both neurological surgeons and general surgeons. In the 800 cases reported to the author the management was chiefly by general surgeons.

TABLE 2

FROM CHART FOR SKULL FRACTURE EXHIBIT SHOWN IN 1934

Date and Source	No. of Cases	Mortality Rate
1927-1934 Mock's Series	171	19%
1928-1934 Collected Cases	800	26%
1928-1934 Hospital Records	1283	39%

What are the causes of death in these patients during the period between the second and the seventh days?

The great majority die from gradual respiratory and cardiac failure due undoubtedly to the cerebral damage. The next commonest cause for death is pneumonia. Meningitis in my experience causes less than 5 per cent of the deaths. Multiple injuries, diabetes, syphilis, alcoholism and old age are all marked contributing factors to the death rate.

In regard to meningitis, meticulous care of the bleeding ear, avoiding antiseptic spraying or irrigation of the wound through any of the

sinuses and early, as soon as shock is over, débridement and cleansing of the compound fractures with loose closure of the wound will prevent many from developing this disease. When it does develop, early and frequent spinal drainage until the cell count is again practically normal, sometimes necessitating fifteen or more punctures, is the treatment to follow. The use of antimeningococcus serum in these cases is of no value unless the specific organism is the cause.

Don't mistake meningismus, from the irritation of blood the result of a subarachnoidal hemorrhage, for meningitis. When meningismus is present it is likewise an indication for repeated lumbar punctures.

One must always be on guard to detect the pneumonia case especially if the skull fracture patient is in the fifth decade of life or over. These patients, often stuporous or in coma, are left absolutely quiet on their backs for too long periods. Frequent change of position to avoid hypostatic congestion, often the forerunner of pneumonia, is important. The oxygen tent or inhalator is of the greatest value in many cases showing respiratory difficulty. I have seen patients who have been in coma for as long as two weeks and have not eaten a mouthful during that time. Patients have died from dehydration and starvation while in coma. This can be avoided and lives saved by the simple measure of passing a stomach tube and feeding the patient at frequent, regular intervals. Good nourishment is likewise a preventive of many complications including pneumonia.

The more specific therapeutic measures to prevent death from cerebral damage are outlined in chart B. The author has classified his cases in four groups according to signs, symptoms and treatment. These groups will be used to elucidate the therapeutic management:

GROUP I, or 5 per cent of the author's cases with proved skull fractures, required absolutely no other treatment except *rest in bed.* These cases were free of all signs and symptoms except the x-ray evidence.

GROUP II, or 55 per cent of the author's cases, required only general treatment. These were the cases requiring anything from shock treatment to a complete course of dehydration treatment except spinal drainage. All of these cases recovered except eight that died the first few hours in the hospital before any special treatment could be given.

Your attention is directed especially toward the cases requiring dehydration, a form of treatment in which the author agrees with many others. Its early (after shock has been overcome) and proper administration has undoubtedly saved many of these 55 per cent of cases from going over into the lumbar puncture or operative group.

The patient is usually in coma or extremely restless. His pulse and respiration are slowed but not usually below 55 and 16 respectively. The blood pressure, after the shock is over, is usually stable with a tendency for the diastolic reading to hover around 70. Of course in older individuals both systolic and diastolic may be elevated. The temperature is usually above normal, seldom above 101°. If a sedative is required chloral hydrate or sodium bromide per rectum is usually sufficient. Luminal or some similar preparation may be used. Morphine is avoided. It is surprising how many of these cases show a sedative response to the administration of a dehydrating dose of glucose intravenously.

Fifty cc. of a 50 per cent glucose solution intravenously is administered to the patient with the above picture shortly after admission. It helps restore blood volume and thus aids in combating shock. If symptoms persist, this glucose solution is repeated in three to four hours. In addition, a saturated solution of magnesium sulphate, 4 oz., is now given per rectum providing shock is over. Many of these cases regain consciousness shortly after this and some require no further dehydration, while others will show periodical signs of returning increased intracranial pressure. To these, the dehydration methods are again applied.

A few of these cases will remain between coma and consciousness and will have persistently slowed pulse and respiration and often a lowered diastolic pressure. For these, the glucose solution and magnesium sulphate per rectum are administered every six to eight hours. Whenever dehydration is used, the fluid intake is limited to 20 or 30 oz. each twenty-four hours. If, in addition to this limitation of fluids, glucose and magnesium sulphate are repeatedly necessary, one must guard against overdehydration with its increased temperature, its restlessness, its headache and phenomena easily mistaken for cerebral damage symptoms. In 1931 the author pointed this out and offered the plan of easing up on the dehydration every forty-eight hours in order to *strike a balance.* More recently, Temple Fay has shown the need of guarding against overdehydration. Many cases have been carried along to recovery by this plan of dehydration treatment persisted in for two or more weeks. It is usually unnecessary to give other than the magnesium sulphate per rectum or by mouth once a day after the first week. Quiet surroundings, avoiding discussion of the case with the patient, good nourishment and rest in bed for three to four weeks complete the hospital care of this group. The period of convalescence will vary from six to twelve weeks.

GROUP III. Thirty per cent of the author's cases fell in this group, which is known as the Lumbar Puncture Group. It includes the

more serious skull fracture cases who, because of persistent coma; persistently slowed pulse and respiration, usually below 55 and 16, respectively; extreme restlessness; persistent, severe headaches; a lowered diastolic pressure, frequently down to 48, and any other signs suggestive of persistent increased intracranial pressure, do not yield to the usual dehydration procedures.

It requires the closest observation to detect the difference between the signs and symptoms due to cerebral edema and other causes of a milder form of increased intracranial pressure and the signs and symptoms of a threatened medullary compression due to more severe causes of increased pressure. A lumbar puncture done early, before medullary compression has developed, is often extremely spectacular in its results. Patients will frequently come out of a deep coma a few moments later and even converse with the doctor or nurse. In many of these cases, the puncture is only required once, if the ordinary dehydration, above described, is persisted in.

Cases which have developed deeper coma; rapid, shallow respirations, with no grunt; or Cheyne-Stokes respiration and a rapid, bounding pulse, have definite signs of medullary compression. The lumbar puncture should have been performed before this stage was reached, and certainly must be performed now. It is in these cases that this therapeutic measure is life-saving, although many of these patients die in spite of this effort.

The importance of the pulse and respiration rate and of the blood pressure as a guide to treatment shows the necessity of hourly charting these, especially during the first day.

The lumbar puncture must be repeated if the signs and symptoms, which first indicate its need, recur or persist. In the case of a patient who remains alive for a few days, but is going downhill in spite of lumbar punctures and dehydration, one must search constantly for the least sign of an indication that operative intervention is needed. In the absence of all such focal signs, and where lumbar puncture has been thoroughly tried, it may be necessary to resort to an exploratory subtemporal decompression. Many lives are lost by persisting in lumbar punctures when indications for operative treatment are staring one in the face. (See 3 in chart B.)

GROUP IV, or the Operative Group: Ten per cent of the author's cases have fallen in this group. This may be a little high for the average run of cases, because several of these operations were performed in other hospitals when called in consultation.

The definite indications for operative intervention in skull fractures are the following:

A—Definitely depressed fractures. Many slightly depressed fractures give no symptoms and do not need operations.

B—The compound depressed skull fracture, often with brain substance exuding from the wound.

C—Middle meningeal hemorrhages. Often these extradural hemorrhages are contracoup to the site of injury. Rarely, but it happens, the paralysis is on the same side as the clot. When such a phenomenon is present, and especially with the site of fracture, shown by x-ray, opposite the site of the clot, the surgeon is presented with a condition requiring his keenest judgment. A bilateral craniotomy is usually the only answer.

D—Subdural hematoma. True blood clots are not always found at operation when this diagnosis is made. Frequently the condition is one of an encysted hematoma, containing only débris and serum. It is thought that the increased osmotic pressure within the thinly encapsulated cyst causes an increase of the fluid content, thus accounting for the late symptoms in subdural hematoma. Again, instead of a clot, one often finds a collection of cerebrospinal fluid. Sachs described this condition, explaining that it was due to a laceration in the arachnoid, thus allowing the escape of the fluid subdurally.

E—Subtemporal decompression. As a rule, this operation is now limited to those few cases with persistent symptoms, growing worse, not responding to lumbar punctures and with no definite focal signs. In my experience, it is not indicated in more than 1 per cent or 2 per cent of the cases.

It may be indicated to do the decompression at the site of the fracture. In such an instance, it is more in the nature of an exploratory craniotomy.

A fracture may be so severely depressed that an early x-ray is indicated to evaluate the risk of delaying surgery. When the depression is extensive and the condition of the patient is such as to indicate that this pressure must be relieved, it may become necessary to operate even within the first few hours. Fortunately, such a situation seldom arises, but when it does, the operation may be life-saving.

The operative rates in the various sources from which the 2,595 cases were collected were as follows:

A—In the 850 cases reported by surgeons to the author	11.6%	operated
B—In the author's 200 cases	10.0%	"
C—In the 1,354 hospital records studied	3.3%	"

In the reported cases, it was evident that some surgeons were resorting to subtemporal decompression too readily, too early, and with a resulting higher mortality rate.

In a study of the hospital records, many cases were found where lumbar punctures were persisted in when signs and symptoms were present definitely indicating the need for operation. In one of the largest hospitals, the staff was proud of its conservative management of skull fracture cases, judged by its low operative rate. There was a time when the operative rate in skull fractures reached as high as 24 per cent (from author's study of the literature on head injuries from 1910 to 1920). In the following decade, many authors wrote on the conservative treatment of head injuries, stressing the nonoperative treatment. They rendered a valuable service, because the reports in the literature from 1920 to 1928 showed the operative rate reduced to 12 per cent with a corresponding drop in the gross mortality rates reported in these articles from 49 per cent in the 1910-1920 period to 33 per cent in the latter period.

Today it would seem that the pendulum is swinging too far toward this so-called conservatism, and cases which definitely demand operative intervention are being neglected, with a corresponding rise in the mortality rate.

In conclusion, allow me to point out that during the last decade our medical literature has been so replete with controversial views concerning the management of acute craniocerebral injuries that the surgeon caring for only a few of these cases each year is left in doubt as to certain procedures. It has been said in condemnation that 90 per cent of the profession are using dehydration and lumbar punctures in head injuries. My studies lead me to believe that this is true. I am positive that 90 per cent of our profession would not persist in these methods if they were dangerous and of no life-saving value. Controversy concerning these two methods should be replaced by a united effort on the part of all workers in this field so to visualize conditions that the average surgeon in the average-sized town will know when to use dehydration, when to institute spinal drainage, and when to resort to operative treatment.

The high mortality rate from skull fractures can be reduced by observing the commonsense principles herein set forth and by the proper application of general and special therapeutic measures.

DISCUSSION

PRESIDENT LORD: I shall ask Dr. Emery Fitch to discuss this paper first.

DR. EMERY M. FITCH: *Mr. President and Gentlemen*—I am very glad that I was the one chosen from this Society to discuss Dr. Mock's paper, because I have always breathed a sigh of relief when I felt the train pulling out of Chicago and I still had a whole head, and again because Dr. Mock not only represents Chicago, but he represents a vast study of skull fractures all over the country and brings a message to us which we should heed. Another reason that I am glad to discuss this talk of Dr. Mock's is because after going home from the New Hampshire Surgical Club last fall, having listened to Dr. Munro, I felt that something definite should be done in our own little hospital, and other doctors who were there from our town felt the same way. So we corrected at once the bad habit of rushing head injury cases to the x-ray room and rolling them over and over in our effort to get different views of the skull so that we could give a definite report to the newspapers as to whether that person had received a skull fracture. I should like to leave with you this message, that you work with your co-workers and try to head off this pernicious habit of rushing patients in shock to the x-ray room just to get an accurate report to give to the newspapers. Really, it matters little what the newspaper knows about the condition of accident patients with head injuries, and we should treat them for shock before we take them to the x-ray room.

We succeeded in getting an agreement among our men that before they tried to x-ray the head injury cases, or the bad accident cases, whether head injury or not, they would first put them to bed and get them out of the initial automobile shock.

The matter of dehydration is enlightening to us all. It is something we can all do, regardless of whether we are surgeons; it is something that every doctor is now familiar with.

The matter of lumbar puncture, we feel, should be reserved for the time when the patient is not improving. We feel that the lumbar puncture should be checked by manometer reading and should not be persisted in just because we get bloody fluid.

I was glad that Dr. Mock spoke about multiple injuries, because that, also, has been a pernicious habit in many of the hospitals, the doctors feeling that they must at once correct all the injuries, often at the expense of the patient's recovery. The other injuries can wait; we must get our patient out of the initial shock, if we are going to do them any good.

I was also glad to hear him speak of the cases that received a laceration of the brain, without necessarily a skull fracture. The fact that you are not able to read a skull fracture into your picture doesn't mean that you are permitted to neglect treatment of the head injury. You must take into consideration the symptoms and treat the brain laceration.

THE MECHANICS OF DELIVERY*

Especially As It Relates To Intracranial Hemorrhage

BY FREDERICK C. IRVING, M.D.†

Mr. President and Members of the New Hampshire Medical Society:

TO obtain an idea of the frequency of intracranial hemorrhage I have consulted the records for the past ten years of the Boston Lying-in Hospital, as fairly representative of a large institution where about 2,000 infants are born annually. During this time there were 20,827 deliveries with an incidence of intracranial hemorrhage of 1 in 107 births. Since intracranial hemorrhage is not always a fatal disease, not all of these babies died. The diagnosis was made in many instances on the clinical signs alone, and could only be checked anatomically when an autopsy was performed. So far as the actual neonatal deaths from intracranial hemorrhage are concerned they have shown a steady drop from 1932, being 5.2 per 1000 in that year, 3.8 in 1933 and 2.5 in 1934.

A review of 182 autopsies on infants showed that 72, or 40 per cent, died from intracranial hemorrhage. Those who exhibited cerebral or cerebellar bleeding of a degree insufficient to cause death are not included in this number.

Intracranial hemorrhage may be due to trauma, intrauterine asphyxia, or it may be a manifestation of hemorrhagic disease of the newborn. In the minds of many, however, the chief cause is violent or unskillful operative delivery, either by forceps, internal podalic version or breech extraction. While deaths resulting from such procedures are far too frequent, they do not supply the entire casualty list in intracranial hemorrhage. In 40 per cent of our infants who presented at autopsy evidences of bleeding within the skull there were hemorrhages also in other parts of the body. The only logical inference then is that in almost half of these babies the intracranial hemorrhage was due not to trauma but either to asphyxia or to hemorrhagic disease of the newborn. Moreover, 25 per cent of our autopsy material showed petechial hemorrhages elsewhere in the body, but none in the cranial cavity.

The production of petechial hemorrhages scattered throughout the various organs is a recognized pathological picture in asphyxia of the newborn. It is the duty of the obstetrician to recognize during labor the signs of approaching asphyxia and to take the proper steps to avoid the fatal results which may ensue to the infant. Auscultation of the fetal heart beats should be carried out at least every half hour during the first stage of labor and at least every five minutes after the os is fully dilated and the expulsive phase has begun. Any marked slowing of the heart rate, or any marked acceleration, or any combination of the two, or an irregularity in rhythm, is suggestive of disturbance in the gaseous exchange with the mother. Fair criteria of threatened asphyxia are consistent rates above 180 or below 100. Of the two a low rate is more important than a high one.

In the first stage of labor, especially if the membranes are unruptured, moderate alterations of the fetal heart rate are seldom of significance and do not call for interference. A notable exception, however, is prolapse of the cord. In the second stage of labor marked alterations in the fetal heart rate, especially if accompanied by the passage of meconium, are of grave importance and indicate the termination of labor by the most appropriate and safest method which, in the majority of cases, is by the low forceps operation. The passage of meconium stained liquor amnii, on the other hand, is of no pathological importance.

Asphyxia may also result from a prolonged and unprogressive second stage of labor, the fetal head being thrust for many hours against an unyielding perineum. In such cases a liberal episiotomy will often result in prompt expulsion of the fetus, although in some instances it is necessary to supplement this small operation by the use of low forceps. The second stage of labor should never be made an endurance contest between the pelvic floor of the mother and the cerebral circulation of the infant.

The second cause of intracranial hemorrhage is hemorrhagic disease of the newborn. While the obstetrician can do nothing to prevent hemorrhagic disease he has the first opportunity to diagnose the condition and to begin its treatment. Bloody stools or vomitus may be present, as may petechial spots on the skin surface. A prolonged bleeding and clotting time are sometimes, but not always, found. Transfusion usually is of benefit, but the intramuscular injection of blood usually exerts its influence too late to be of any aid.

The third cause of intracranial hemorrhage is trauma. Trauma may result from an attempt to deliver an infant which is too large through a pelvis which is too small. However, cephalopelvic disproportion is not the most common cause of trauma, since pelvic contraction occurs only about once in every 300 cases in New England. By far the most frequent source of fetal

*The second in a series of papers presented in the Symposium on Obstetrics at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 7, 1935.

†Irving, Frederick C.—William Lambert Richardson Professor of Obstetrics, Harvard University Medical School. For record and address of author see "This Week's Issue," page 653.

injury is delivery through an undilated cervix, and far more babies lose their lives from this cause than from attempts to overcome bony obstruction. A mistaken notion that the interest of the mother or child is conserved by delivery during the first stage of labor, or, in many cases, the haste of the attendant to terminate labor for his own convenience is responsible for a large number of dead infants, and a few idiots among these which survive, to say nothing of a large amount of maternal wreckage which in time draws these unfortunate women to the gynecologist. In the end patience will best serve the mother and child. The use of analgesic drugs in labor will assuage the pains of labor and make the attendant more willing to await the workings of nature. It is significant that the last three years, which have shown almost a 50 per cent drop in the neonatal stillbirth rate at the Boston Lying-in Hospital, covers the period of the routine use of hypnotic drugs. At this point it is well to state that the barbiturates should never be used in cases that are confined at home, as the excitement that is sometimes caused may result in injury to patients that are left unattended.

The infant mortality rates following the different varieties of forceps operations in our clinic have been:

Low forceps	1.6 per cent
Mid forceps	7.0 per cent
High forceps	20.0 per cent

It is therefore evident that the danger of a skillfully performed low forceps operation is slight, but that the risk increases steadily the higher is the station of the head in the pelvis. In the years from 1931 to 1934 inclusive there were only five high forceps operations among 8,617 deliveries. One of the infants died. It is therefore evident that the high forceps operation is fast becoming obsolete.

The fetal death rate in our clinic in internal podalic version over a number of years has been 16.7 per cent, which approaches that of high forceps. From 1932 to 1934 inclusive there were performed fifty-three versions and extractions in 8,617 deliveries with the loss of fourteen babies, a mortality of 26.4 per cent. Moreover, over half of the patients referred to the hospital with ruptured uteri have received their injury as the result of this method of delivery. As a potential menace to both mother and infant this exceedingly dangerous operation takes first rank and its routine and indiscriminate employment, as is advocated in some quarters, cannot be too strongly condemned.

Although not associated with intracranial injury, it is pertinent to make some mention of brachial paralysis. This injury may be of two types: (1) a paralysis of the shoulder girdle and (2) true brachial or Erb's palsy. A paralysis of the shoulder girdle may be due to pressure of the forceps blades in vertex deliveries or by the fingers in releasing the arms in breech extractions. There is a flaccid paralysis of the arm and shoulder girdle which is usually not evident until from twelve to twenty-four hours after birth. Since the signs are due to hemorrhage into or about the nerves, and not to a parting of their trunks there is no permanent damage, and recovery may be expected within a month. Erb's palsy, on the other hand, is caused by traction on the head to effect delivery of the shoulders in head first presentations or by extreme extension and elevation of the shoulders in breech extraction. Actual tearing of the nerve trunk occurs, and the paralysis is immediate and permanent. Unfortunately, even the results of orthopedic surgery in such cases leave much to be desired.

Brachial paralysis of both types has occurred once in 1,000 deliveries in our clinic. No traction should ever be applied to the head to release impacted shoulders. Suprafundic abdominal pressure applied by an assistant will suffice in most cases. When it fails, a hand should be inserted into the birth canal and the infant's arm swept downward and out across its chest. Only by the exercise of these precautions can one avoid this distressing accident, which not only unfits the individual to earn his living by the use of his hands, but also in many instances produces a marked psychological effect upon him.

THE PREVENTION OF PUERPERAL INFECTION*

BY FOSTER S. KELLOGG, M.D.†

IT was with much pleasure that I came to Manchester with this assignment.

I do not purpose to present an academic discussion of puerperal infection. My intention is to take up certain phases of the subject that have been especially brought to my attention and by summarizing these to point out practical ways to reduce maternal mortality and morbidity from this condition.

Roughly, twenty-five per cent of maternal mortality is accounted for by sepsis. It is not possible to figure accurately the morbidity. Students of the subject seem to believe that this state of affairs has not improved in the last three decades.

For the purpose of this discussion puerperal

*The third in a series of papers presented in the Symposium on Obstetrics at the Annual Meeting of the New Hampshire Medical Society at Manchester, May 7, 1935.

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sepsis in all its manifestations may be divided etiologically into these three groups:

First: Wound infection at the time of labor or delivery.

Secondly: Autogenous infection, manifesting itself prior to, in, or after labor.

Thirdly: Infection during the puerperium by organisms transmitted to the genitalia by an unsterile digital contact; or by laughing, talking, sneezing or coughing of an attendant carrying pathogenic organisms in the nasopharynx during so-called "perineal precautions"; or by the same mode of transmission from infected appliances or applications to the region of the perineum.

It is the widely accepted opinion at the present time that the usual offending organism in puerperal sepsis is the streptococcus and that some form of hemolytic streptococcus is usually to blame for the severe cases.

Dr. John T. Williams of the Boston City Hospital has shown that there is an absolute correlation between high points in the Massachusetts State Department of Health index of respiratory disease and severe epidemic puerperal sepsis; the latter following the former in its appearance by thirty to sixty days.

Because the three divisions overlap and for the sake of brevity I shall enumerate, without reference to this grouping, such practical measures as may be expected in the light of our present knowledge to reduce the incidence of puerperal sepsis.

DIGITAL ASEPSIS AT TIME OF DELIVERY

This cardinal principle of obstetrical technic is usually assumed. We saw in consultation not so long ago a woman, dying of puerperal sepsis. The man who delivered her had a septic cut on one finger. This is an extreme example. The hands of a general practitioner, constantly in contact with scarlet fever, erysipelas, tonsillitis, taking throat cultures are not far different as potential infecting agents save that with an intact skin a ten-minute scrub and sterile gloves prevent it. That carefully timed scrubbing is actually efficacious in practice in establishing a safe degree of relative digital sterility was shown to me long ago. When I was a house officer at the old Lying-in Hospital it was a duty, since happily given up, of the Junior man to wash out the uterus of each case in the septic ward once a day; it was another duty to deliver the normal cases in the hospital. By prolonged scrubbing before the septic wash and after it, and before the clean delivery, cross infections were avoided. It has been my practice for a long time to double scrub before a clean delivery if I have had a recent septic contact or if I have a nasopharyngeal infection at the time of delivery.

PREPARATION OF PATIENT FOR DELIVERY

It has been our experience that soap and water scrub with alcohol or other solution, or that any of the accepted so-called dry preparations of the skin and vulva are satisfactory. We do not believe in ante-partum vaginal douches or "dry" solutions in the vagina itself and we have never felt that the instillations of one or other germicide into the vagina were valuable. It is believed by some investigators that organisms harbored in the patient's vagina are seldom pathogenic to the given patient under ordinary circumstances of delivery and if they are pathogenic are usually only mildly so. It is also believed that the patient's own intestinal flora are harmless to her, except when she herself has a respiratory infection. In this event they may be very virulent in the uterus and extraordinary precautions against fecal contamination in labor and throughout the puerperium must be practiced.

POSSIBLE DANGERS OF CATHETERIZATION

Catheterization when necessary during labor must be routinely performed, and great care must be exercised in the performance for reasons familiar to you. It is, however, not of ante or postpartum catheterization I wish to speak. At the time of delivery a patient may have an infecting organism in her kidney pelvis with or *without* clinical pyelitis. It is customary to prepare the patient, then catheterize her and with little further preparation deliver her, perhaps with forceps. I have seen a patient with a streptococcus urine thus infected per vaginam with the forceps. We are all, I think, too careless in the assumption that urine through and around a catheter at time of delivery is necessarily sterile. This is one example of true autogenous infection, and an avoidable one.

TYPES OF DELIVERY THAT INVITE INFECTION

The commonest of all causes of infection at time of delivery is the secondary factor of mistimed and unnecessary operative delivery. This is an avoidable cause of many septic deaths and much septic morbidity. There is no such thing as the so-called "dilatable cervix" from an operative point of view. Either there is a cervix or there isn't. If there is, no operative procedure from below should be undertaken. By avoiding indiscriminate vaginal examinations, by patience and the wise use of modern obstetrical analgesics such as pentobarbital and scopolamine, rectal ether and paraldehyde almost every cervix will in time dilate fully, and a very large proportion of these cases will deliver normally or by moderate pressure from above, with episiotomy; or come to perineal forceps with the head in sight. If a given patient fails to dilate her cervix completely after enough time has been allowed, she may safely have a

cesarian section if the case had been followed by rectal examination. I do not believe in prolonged labor in the face of frank disproportion which may be detected by examination, nor do I believe in too prolonged labor against the rare intractable cervix, but I do believe that, since the cardinal pathology of delivery infection is a septic focus in the split cervix, practically no operating should be done from below unless the cervix is fully dilated.

CONDITIONS CONTRIBUTING TO INFECTIONS

Certain general conditions in the patient and certain conditions specific to pregnancy seem to tend to increase the likelihood of infection. These are toxemia and nephritis, pyelitis, heart disease, and anemia, either that prone to occur in pregnancy or produced at delivery by blood loss as in placenta praevia and separation of the normally implanted placenta. Methods of delivery calculated to keep blood loss at a minimum, especially a deliberate and careful conduct of the third stage of labor, especially in these complications of pregnancy, is worth while. It is my opinion that even in well-conducted maternity centers too little attention is paid to the hemoglobin, red blood count and hematocrit. In the hospital after delivery each patient should have a count and hemoglobin. It is my belief that three million or less reds at this time calls for a transfusion, and that if the patient shows any elevation of temperature a transfusion should be given even though the count is three to three and one half million, and in some instances even if it is higher.

MASKING IN OBSTETRICS

It is considered that most epidemics of hospital sepsis and that most severe puerperal sepsis is the result of infection from the nose and throat. Very occasionally this appears to be autogenous, usually it is exogenous. It may take place at the time of labor or any time in the puerperium. It occurs equally by direct transmission of mouth and nose droplets, or by indirect digital transmission of the same. So it must be reasoned first that digital asepsis of a high order is as necessary for contacts—usually nurses giving perineal precautions—throughout the puerperium as in the delivery room. This is obviously an administrative problem. Incidentally it has often been found in puerperal hospital septic outbreaks that a failure to follow medical orders to the letter by the administrative heads of a hospital is completely or partially responsible for the epidemic. Secondly: Masking of the most effective type used faithfully over the nose as well as the mouth—and not around the neck, a place I have frequently seen the mask worn especially by pupil nurses—is absolutely essential both in the labor and delivery rooms and throughout the puerperium during contacts. It has been shown experimentally that a mask becomes wet on the outside in about ten minutes and transmits organisms to petri dishes some distance away if the subject of the experiment talks or coughs. Therefore it is my practice to double mask if I have a cold at time of delivery and in any event to conduct delivery or abdominal section without talk. If orders or discussion or coughing are imperative, the face should be turned away for the time being. If a mask becomes visibly moist on the outside another may be tied over it. Nurses should be made to carry out perineal precautions in silence. It is usually true that nurse-borne epidemics of throat-borne sepsis may be attributed to an individual who has had a fairly recent *acute* upper respiratory attack sufficient to put her off duty for a few days. Hence such nurses must not be allowed to return to obstetrical duty until cultures show the throat and nose negative for hemolytic streptococci. The wider use of throat cultures should be used in so far as it is practical, but dry masking and silence will prevent much present-day severe puerperal infections from the nursing service.

EXPERIENCE WITH OTHER METHODS OF INFECTION

We have seen two patients die infected apparently by perineal icebags supposedly sterilized after contact with a case of streptococcus uterine sepsis of undetermined origin who recovered, showing the commonly seen increased virulence of the organism as it is passed through other victims. For this reason we have abandoned the use of the perineal icebag entirely. We have some reason to believe that solutions of one sort or another used to paint perineal stitches for comfort or supposed antisepsis have transmitted infection so the use of these we have abandoned. We use opium suppositories ½ to 1 grain in the rectum for perineal comfort.

We have seen two cesarian sections die of peritonitis, operated the same summer week in the same institution with the same rare infecting organism apparently from dropped perspiration of one man who took part in both operations. No other sepsis was present at the time. We therefore set the rule that one nurse in the operating room is assigned to "watch for and mop sweat" on sight—irrespective of the momentary feelings of the operator; and make her entirely responsible for this. It is not reasonable to expect the surgeon and others in the operating room to do "sweat detection" in addition to other duties.

TREATMENT

The scope of this paper calls for little or no comment in respect to treatment of puerperal infection. In the future and where at present available, "immune transfusion" gives the only specific hope ever offered to these patients except the difference in virulence of the various

organisms and the patient's own individual resistance. Simple transfusion, especially in relation to the blood picture, often repeated, general supportive measures including rest, and the drainage of frank pus when it reaches the surface per vaginam or otherwise are at present our only remedies. Therefore prophylaxis should be our aim.

SUMMARY

Digital asepsis in labor and throughout the puerperium, dry masking and silence throughout the same periods, cultural watching of nasopharyngeal suspects, *an abject respect for the integrity of the cervix by the physician*, care of the potentialities for infection at time of delivery from the urinary tract, an accurate knowledge of the blood picture after labor and at other times if it seems wise, appropriate and immediate correction of this picture if it is a poor one, abandonment of perineal icebags and "perineal solutions," and a scrupulous regard of operating room technique including the "sweat risk" constitute the sum of this prophylaxis. Carried out in careful detail I believe a highly creditable reduction in maternal mortality and morbidity from puerperal sepsis can confidently be expected.

MISCELLANY

LEONARD JARVIS, M.D.

Dr. Leonard Jarvis, for fifty-two years a practicing physician in Claremont, as were his father and grandfather before him, died on January 28, 1936, at his suite at the Hotel Moody. He was eighty-three years old and for several years had been in failing health, continuing, however, up to within a few days of his death, the practice of the profession to which his life had been devoted. Taken critically ill the previous week he failed to rally and death claimed Claremont's best loved citizen.

Dr. Jarvis was born in Claremont on July 29, 1852, son of Dr. Samuel and Sarah Jarvis and grandson of Dr. Leonard Jarvis. He was graduated from Kimball Union Academy in 1869, from Dartmouth Medical College in 1873 and received his medical degree from Harvard in 1882. After serving internships in Boston and Providence, he returned to Claremont in 1884 to engage in practice there.

On June 25, 1893, he married Miss Mabel Howard of Providence, R. I. To them were born two children, a daughter, Caroline, who died in 1906 and a son, Samuel Gardiner Jarvis.

Dr. Jarvis was a member of the American Medical Association and of the New Hampshire Medical Society, being publicly cited by the latter organization in May, 1932, as one of the five Granite State physicians who had served continuously for half a century. Two years later, on May 16, 1934, he was the recipient of the Society's highest award, the fifty-year gold medal.

Dr. Jarvis was for many years a director of the Claremont National Bank. He was for thirty-four years a warden of Union Church at West Claremont and, in April, 1934, was named senior warden-emeritus of this church.

Dr. Jarvis is survived by his widow, Mrs. Mabel Jarvis and son, Samuel Gardiner Jarvis, of Quincy, Massachusetts.

THE APPOINTMENT OF DR. MILLER

Dr. Ralph E. Miller, assistant professor of pathology at the Dartmouth Medical School, has been appointed assistant dean of the school and promoted to the rank of associate professor of pathology. He has been granted sabbatical leave for the second semester of the current academic year in order to study pathology under Professor Pick in Berlin.—*Science* **83**: 228 (Mar. 6) 1936.

HOSPITALS

The Woman's Board of the Littleton Hospital, aided by the Baldwin Fund, purchased, early in December, an oxygen tent and a portable x-ray machine complete with a fluoroscope.

NURSES

The quarterly session of the New Hampshire Graduate Nurses' Association was held in Manchester, December 11, 1935. Under the direction of Mrs. Alma VanPelt, Superintendent of Nurses at the Elliot Hospital, Manchester, the League of Nursing Education met separately during the morning to consider general business and to listen to a reading of nursing school records.

The principal speaker of the afternoon was Miss Ella E. McNeil of New York City, Assistant Director of the National Organization for Public Health Nursing, who discussed "Broader Aspects of Community Responsibility for Nursing."

Miss Rose Griffin, President of the Association and Superintendent of the Mary Hitchcock Memorial Hospital in Hanover, presided at the afternoon session. The New Hampshire Board members of the organization for Public Health Nursing met with Miss Ruth Whitcomb of Concord.

Dr. Warren Butterfield of Concord spoke to the delegates on "The First Aid and Accident Prevention Program of the Red Cross."

The officers of the organization are Miss Rose Griffin, President; Miss Vernice Patterson, 1st Vice-President; Miss Alice E. Russ, 2nd Vice-President; Mrs. Melaine R. Proulx, Secretary and Miss Loretta Landry, Treasurer.

The quarterly meeting of the Executive Board of the Keene District Nursing Association was held January 9, 1936. The total number of visits during the last quarter was 1,287. Of these there were 598 medical; 229 surgical; 176 obstetrical; 164 newly born; 120 welfare. The Association has recently purchased an auriscope for the use of the

nurses in examination of babies and young children.

The monthly meeting of the Portsmouth Nursing Association was held January 2, 1936. The Association enters upon its thirty-first year.

CANCER

A series of radio broadcasts, sponsored by the State Cancer Commission, have been given over Station WFEA, Manchester, recently. Speakers were Dr. James W. Jameson, Concord; Dr. Deering G. Smith, Nashua; Dr. Emery M. Fitch, Claremont; Dr. Harold J. Connor, Concord; Dr. Clifton S. Abbott, Laconia; Dr. Howard N. Kingsford, Hanover; Dr. Walter H. Lacey, Keene; and Dr. Alfred J. Leary, Manchester.

PERSONALS

Dr. Melba Stewart Perley of Laconia spoke on January 23 to the Mothers' Department of the Lakeport Women's Club. She based her remarks upon her experiences at the hospital in the x-ray department, illustrating her points with x-ray pictures of children.

Dr. Ezra A. Jones of Manchester was the guest speaker recently of the Laconia Rotary Club and the Keene Rotary Club, speaking of his work among crippled children.

Dr. Walter H. Lacey of Keene gave an instructive talk on "Carcinoma" before the Keene Fortnightly Club, January 11, 1936.

Dr. Robert B. Kerr of Manchester was elected President of the New England Tuberculosis Association at its meeting at the Hotel Statler, February 7. Dr. Kerr is Medical Director of Pembroke Sanatorium and Executive Secretary of the New Hampshire Tuberculosis Association.

MEETINGS

Members of the new State Commission for the Study of Occupational Diseases in New Hampshire held their first session on Wednesday, December 12, 1935 at the State House. Members of this Committee are Dr. Robert J. Graves, Concord, Chairman; Eugene J. O'Neil, Concord, Secretary; Dr. Emery M. Fitch, Claremont; Peter Tsiales, Manchester; Daniel Feindel, Berlin; Frederick Graf, Keene; Major A. Erland Guyotte, Peterborough; Charles E. Greenman, Hampton, and Dr. David W. Parker, Manchester.

The Belknap County Medical Association sponsored a skin clinic on January 17. Dr. R. E. McDonnell of Yale Medical School addressed the clinic in the afternoon, illustrating his talk with slides.

The annual meeting of the Hillsborough County Medical Society was held at the Derryfield Club in Manchester on November 15, 1935. The following officers were elected to serve for the year: Dr. Henry H. Dearborn, Milford, President; Dr. Charles F. Nutter, Nashua, Vice-President; Dr. Deering G. Smith, Nashua, Secretary-Treasurer. Dr. Dudley Merrill of Boston spoke on "Dangers Inherent in the Clinical Diagnosis of Cancer."

The Belknap County Medical Society held a meeting at the Laconia Tavern, February 11, 1936. The speakers were Dr. Clifton S. Abbott, President of the New Hampshire Medical Society, and Dr. Carleton R. Metcalf, Secretary of the New Hampshire Medical Society. The program was in charge of Dr. Laura Jacques.

VERMONT STATE MEDICAL SOCIETY

THE CAUSES OF SUDDEN BLINDNESS*

BY ARTHUR J. BEDELL, M.D.†

WHEN a patient suddenly goes blind he usually consults his family physician before going to the ophthalmologist.

The causes of rapid impairment of sight are numerous. Many are easily recognized and often prompt treatment restores function. At other times the blindness is part of a constitutional condition for which little or nothing can be done.

A differentiation should be made of partial and complete loss of sight. To do this best we must adopt a reliable method for determining the degree of visual loss. This examination is divided into two parts; one the central vision and the other the field of vision. The general practitioner can very readily and accurately determine the latter by taking the finger field.

Acute congestive glaucoma is one of the most frequent, most serious and yet most easily diagnosed causes of sudden blindness. The eye condition is so frequently masked by the acute gastrointestinal upset that the picture should be so indelibly impressed upon the minds of all physicians that no case is neglected. In glaucoma one or both eyes present an intense cyanotic congestion of the globe and conjunctiva. The cornea is hazy, the anterior chamber shallow, the pupil ovoid and dilated with a definite increase in the intraocular tension. This last is easily diagnosed by palpation. By placing

*Read at the Annual Meeting of the Vermont State Medical Society at Rutland, October 18, 1935.

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the index fingers in apposition under the upper margin of the orbit a delicate ballottement indicates the degree of resistance. When the eyeball is hard and it is not possible to dent it, increased intraocular tension is diagnosed. In every case of gastrointestinal upset which occurs in an adult the eyes should be most carefully inspected for if acute congestive glaucoma is overlooked, the patient will eventually become blind, although after a variable time the eyeball whitens, the pupil remains widely dilated and some vision may remain but only for a comparatively short period. The process further continues until finally the eye becomes so painful that enucleation is necessary. As soon as the diagnosis is made the patient should be given a hypodermatic injection of morphin, without atropin; eserin 1 per cent solution instilled in the eye, hot compresses applied and arrangements made for an early operation. Delay is dangerous. The operation of choice is an iridectomy and incidentally if you are not doing special work, I urge you to transfer your patient with acute congestive glaucoma to a competent ophthalmologist, not only to protect the patient, but also to save you many regrets for the treatment of glaucoma is an extremely technical one.

Another type of glaucoma comes on so insidiously that there is a steady, imperceptible loss of sight which is frequently not recognized by the patient until there is a very great impairment. In this type the interior of the eye must be examined, the field of vision must be frequently charted on the perimeter and the intraocular tension not only measured by gross palpation but also by the exact instrumental means. This form must be treated with the greatest skill, sometimes eserin and pilocarpin will retard the process, at other times operations are indicated.

Vascular accidents cause the largest group of cases of rapid failure of sight, and although we may become suspicious of a blood vessel disease, we cannot be certain of the diagnosis until an ophthalmoscopic examination is made. Most of our patients have hypertension but some have low pressure, therefore, the blood pressure reading must not be considered as making the diagnosis but only pointing the way.

Two lesions frequently encountered are sudden closure of the central artery of the retina, which may come from an embolus, thrombus or endarteritis, and the rapid occlusion of the central vein. The fundus picture of the artery obstruction is diagnostic. There is widespread edema. The arteries are either narrow or closed, the veins are small and the macular region red. There is very little that can be done for this sudden plugging of an artery. We massage the eyeball, administer vasodilators by mouth or give amylnitrite by inhalation. Opening the anterior chamber has been proposed but I have never seen any good result from this dangerous procedure and, therefore, discourage its use. At times there is canalization of the thrombus with some return of vision. Occasionally there is an auxiliary blood vessel system which furnishes blood to the macula and central vision is preserved.

Thrombosis of the central retinal vein may be complete or limited to one branch. Obviously, the amount of visual loss depends upon the extent of the vein disease. In many cases of partial thrombosis the retinal blood flows over the macular region and the patient complains of blindness. There is often a very considerable improvement. In some cases the blood cells fill the filtering angle of the eye and secondary glaucoma develops causing total blindness and often necessitating the removal of the painful globe.

Complete thrombosis of the central retinal vein may or may not be an evidence of a general vascular disease, and a very exhaustive clinical investigation may fail to disclose the reason for the vein occlusion. The blindness is sudden and unaccompanied by any external evidence of disease, or by pain. The fundus picture is characteristic, the veins are large and distended, there is some retinal edema, many hemorrhages and frequently white patches. The absorption of the blood and the restoration of circulation does not follow any definite order and subsequent changes are bizarre. Treatment is directed to any discoverable body ailment and if the intraocular tension is increased, attempts must be made to control it. Again I repeat that it should be distinctly remembered that both of these serious vascular accidents are found in hypotension as well as hypertension.

Arteriosclerosis of the fundus is so well known and so clearly described by words and photographs that little need be said regarding the various changes which constitute the general picture. A patient with arteriosclerosis may speak of sudden blindness, although in reality the examination will show that he has a full peripheral field of vision and has only lost the central part. This, of course, means that the patient is unable to read. The disease is usually found in those who are senile, and by senility I do not necessarily mean age. It is caused by an obstruction in the perimacular vessels with a hemorrhage in the choroid, cystic degeneration of the macula, and the end result is a pale partially depigmented scar. It is usually bilateral, although one eye may be involved long before the other. Little or nothing can be done to alleviate the symptoms. The patient is always happier if he understands that he will never become blind as a result of the disease. There is an arteriosclerotic optic atrophy. Fortunately,

this is uncommon for it means complete loss of vision in the eye that is involved.

When the retina becomes detached from its choroid base there is always a partial loss of visual field. Some detachments are spontaneous and develop without any discoverable reason. On the other hand the retina may be separated as a result of a direct blow upon the eyeball or the stretching of the globe in high degrees of myopia.

An intraocular growth may push the retina forward. Occasionally the separation is caused by nephritis, particularly that which accompanies pregnancy. Examination in the primary type shows that the gray retina moves as the eye changes position. If a choroidal tumor is present the retina may be adherent to the growth or there may be so much fluid surrounding it that at one stage the retina waves as it does in the subretinal fluid detachment. The diagnosis of detachment of the retina is not difficult but the differentiation between the secondary and primary types is sometimes most confusing and the patient must be observed for some time before an accurate diagnosis can be given. We have three diagnostic methods which are of considerable help in such cases. One is the slit-lamp; an intense, brilliant beam of light is thrown into the eye which is then examined with a binocular microscope. Another is serial stereoscopic photographs of the fundus and the third is transillumination. If the detachment is so located that a small lamp can be placed behind it, the light is readily transmitted through a simple detachment, but where there is a tumor the dense growth prevents the light from being seen in the pupil.

The detachments in acute nephritis and in pregnancy are usually so thick that there is little motion of the retina. They are often bilateral and the general condition of the patient is sufficient to guide one to the correct diagnosis. The retina usually becomes spontaneously reattached in these diseases.

Detachment of the retina has for years been looked upon as a hopeless condition and, although patients have been subjected to all sorts of rest and dehydration methods, little success has attended the physician's efforts. But now, thanks to the work of many ophthalmologists scattered over the world, and especially to the late Gonin of Lausanne, Switzerland, operations can be performed upon certain types of detachment. The hole in the retina can be seen and then sealed by means of high frequency or galvanic electricity. This is a technical operation, but the restoration of vision in such a patient gives the ophthalmologist great satisfaction. It is important to remember that if a patient with detachment is to recover his vision by operation, the operation must be performed before the retina has lost its function. When the retina is displaced by a growth, the eye should be enucleated, for usually the tumor is a sarcoma of the choroid which has a predilection to metastasize in the liver and orbit.

Impure alcohol, wood alcohol and various drugs can cause sudden and complete bilateral blindness which is often ushered in by an acute gastrointestinal upset, sometimes with coma, and unless the eyes are carefully examined the vision may be totally destroyed before the patient becomes conscious. Here again prompt action is imperative. The stomach should be washed out several times a day, saline purgatives administered, lumbar puncture immediately performed and a small dose of salvarsan injected intravenously. If the patient survives, strychnin should be given internally and positive galvanism applied to the eyes. Destructive blindness has been caused by large doses of quinine and more recently by the application of thallium in the form of a depilatory. Since the introduction of dinitrophenol many patients have presented with beginning blindness. In the cases that I have seen the earliest changes have been in the lens, a curious widespread opacification. Some have progressed to complete cataract, others have remained immature.

Partial blindness follows the prolonged use of tobacco in those who are susceptible to nicotine poisoning. There is a definite loss in the field of vision and the history of tobacco consumption. By the inhibition of tobacco and the use of strychnin, many are cured.

There seems to be decidedly less use of quinine sulphate as an abortifacient. I have seen a patient after the ingestion of a cupful of the drug. The uterus was emptied but the patient remained totally blind, the nerve atrophic and the retinal vessels small.

With the more extensive use of the automobile, and the increased manufacture of storage batteries, lead poisoning is more prevalent than formerly. In some parts of the world, where they still use lead pipes, children are frequently affected, the optic nerves are involved and the sudden blindness almost total. Lead poisoning is often difficult to diagnose. Not only must we have a history of the occupation of the patient but the diagnosis must be confirmed by chemical studies. In the cases that we have seen, the removal of the source of the poisoning, change of occupation, the institution of elimination measures such as the drinking of large quantities of milk, the ingestion of potassium iodid and fresh air have resulted in a favorable outcome.

Sudden blindness may come from an infection of the nasal sinuses. The patient has a head cold and notices that his vision is at first blurred and then becomes suddenly lost.

One of the earliest signs of multiple sclerosis is a rapid loss of sight in one or both eyes. As this disease has periods of remission, a patient

may be treated for a time and as a result of constitutional treatment show a very great improvement but, unfortunately, eventually he becomes sightless.

Injury to the head may cause blindness either by a hemorrhage in the optic nerve sheath, a hemorrhage in the orbit, a fracture of the orbit or a severance of the nerve itself. Every case of trauma to the head should be thoroughly examined by a competent ophthalmologist.

Puncture wounds of the eyeball may cause sudden blindness either from infection or primary destruction. Compression of the eyeball may produce a hole in the macula or a rupture of the choroid. These conditions can only be discovered when the ophthalmoscope is correctly used. A severe compression of the chest can result in blindness by extravasation about the optic nerve leading to subsequent atrophy of it. The degenerative changes in the retina, choroid and optic nerve may cause a rapid loss of reading power and the patient speaks of blindness. This defect is partial and can only be diagnosed by the use of the ophthalmoscope.

Tuberculosis, syphilis and focal infections cause much blindness. Multiple small thrombotic hemorrhages in the retina may come from the various blood dyscrasias such as leukemia, or may be an ordinary allergic reaction. If the hemorrhages do not involve the macula, we frequently have restoration of central vision. In diabetes the patient may have a very great increase in the amount of blood sugar without any change in sight. He may on the other hand have a slight increase above the normal with very intense retinal and choroidal destruction, or only a central scotoma.

In chronic nephritis the patient loses sight as a result of retinal degeneration. The changes in the fundus found in this disease, as well as in hypertension, are illustrated by the slides. There may be a marked decrease in sight after exposure to bright light, such as in those who have stared at the sun during its period of eclipse, prolonged exposure in the snow and those who use the arc for welding. Blindness may also follow the use of certain drugs like atoxyl and ethylhydrocuprein. Brain tumors may cause partial or complete loss of vision. And finally, sudden blindness may be complained of by an hysterical patient at which time the physician must use all of his professional skill and native ingenuity to distinguish between it and some of the obscure brain diseases which it simulates.

Sudden blindness is sufficiently common and its causes sufficiently varied to warrant your attention to the problem, so that you may be prepared when the emergency arises. By concerted effort on the part of all of us, much blindness can be prevented and much relieved.*

*Illustrated by black and white and colored photographs of the fundus and external conditions.

SYPHILIS IN PREGNANCY

Summary and Conclusions*

1. A report is made of the effect of treatment on the outcome of pregnancy in syphilitic women. The data show that congenital syphilis is practically a preventable disease. *Its prevention is dependent upon the routine, early and repeated use of the serologic blood test on every pregnant woman and upon adequate early treatment once the diagnosis of syphilis has been made.*

2. A positive blood reaction *during pregnancy* is a serious matter to the fetus. Ten times as many syphilitic children were born when the syphilitic mother's blood was positive during pregnancy as when it was negative.

3. The pregnant syphilitic woman was found to tolerate anti-syphilitic treatment as well as, or better than, the syphilitic woman who had not been pregnant since infection.

4. There is evidence that habitually aborting syphilitic women are capable of producing living, apparently nonsyphilitic children when given specific treatment throughout each pregnancy.

5. Many more nonsyphilitic living children were born when anti-syphilitic treatment was begun before the fifth month of pregnancy than when therapy was delayed. This advantage was increased if the treatment during pregnancy was not only early but adequate, that is at least 10, preferably 15, injections of arsphenamine and appropriate heavy metal.

6. If an early syphilis appears late in pregnancy some treatment begun at this period and continued up to termination of pregnancy, even though it is only a small amount, will be of value in the production of a living child. To those women with early syphilis who were treated after the fifth month of pregnancy only 7.6 per cent of the children were born dead whereas among a similar group of women with early syphilis to whom no treatment was administered during pregnancy the loss of life was 46 per cent.

7. Treatment during a preceding pregnancy is insufficient protection for the present pregnancy even though the syphilitic woman has a negative blood reaction. It is necessary to treat her throughout each pregnancy to insure a living nonsyphilitic infant.

8. The important factors in controlling clinical progression and relapse in the syphilitic woman are the stage of syphilis on beginning treatment and the amount of therapy administered, rather than the pregnancy. The possible exception is the apparent protection pregnancy affords the early syphilitic in avoiding an involvement of the central nervous system.

*Abstract from the February *Bulletin of the United States Public Health Service.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22131

PRESENTATION OF CASE

First Admission. A white married unemployed fifty-three year old Syrian male entered complaining of pain over the sternum.

His illness began eleven weeks previously with dyspnea, a sensation of precordial constriction and wheezing respiration. The onset was gradual and these symptoms persisted with varying intensity for about three weeks. During this time he also had knifelike pains in the small of his back and stated that there was swelling of all parts of his body which persisted for about six weeks. This had subsided before admission and his only complaints then were residual weakness and nocturia.

Physical examination showed a well-developed and nourished man in no acute distress. The fundi showed tortuosity of the retinal arterioles. The heart was enlarged and there was a systolic mitral murmur and an accentuated A_2. The blood pressure was 210/140. The liver edge was just felt.

The temperature, pulse and respirations were normal.

The urine contained a large trace of albumin and an occasional granular cast and red blood cell, but was otherwise negative. Examination of the blood showed a red cell count of 6,300,000, with a hemoglobin of 75 per cent. A phenolsulphonephthalein test showed 60 per cent excretion and the nonprotein nitrogen of the blood was 36 milligrams per cent. There was a normal urea clearance. A Hinton test was negative.

X-ray studies showed dullness of the right antrum with thickening of the lining membrane. There was also slight clouding of the right ethmoid cells. Measurements of the heart were within normal limits. The apex was blunted and the aorta tortuous.

He was discharged in eleven days.

Second Admission, two years later.

He was followed in the Outpatient Department where his urine was found to show a large trace of albumin persistently and his blood pressure remained elevated at about 220/120. Systolic and diastolic murmurs were audible at the left sternal border. Two months before admission a gradually progressive weakness became so pronounced that he was unable to continue his work. There was occasional swelling of the ankles, dyspnea with very slight exertion, and a nocturia of two to three times. An occasional twinge of pain behind his sternum was readily relieved by nitroglycerine. Three weeks before admission he began to suffer from a "tight pain" behind the middle of the sternum following the slightest effort. Rest, nitrites or a warm local application relieved his distress within five minutes. Two weeks later, following an emotional upset, the pain became much more severe and was not relieved so readily even with continued bedrest. Four days prior to admission the pain became quite intense and persistent. He had a queer tingling sensation in both hands and sweat profusely. The discomfort was at first unaffected by hypodermic medication but later he was able to sleep fitfully. Two days later the pain became unbearably stabbing in character and radiated from the third and fourth interspace at the right parasternal line directly to the back. He coughed up some bloody sputum. There was cyanosis and dyspnea which were relieved somewhat by hypodermic injections and dry cupping. Repeated emesis at the onset of this episode subsided in two days and an annoying cough productive of a small amount of yellow sputum developed and persisted. The cough caused considerable discomfort in a region situated a little above the left nipple.

The past and family histories are non-contributory.

Physical examination showed a well-developed and nourished middle-aged man sitting upright in bed complaining of constant precordial pain. There was flattening of the occiput. The fundi were not examined. There was dullness in the right chest from the sixth rib anteriorly and the eighth posteriorly to the bottom. Tactile fremitus and vocal resonance were increased in this region. Fine râles were audible at both bases and there was a pleural friction rub at the right bottom posteriorly. The heart did not appear to be enlarged. The sounds were regular and of good quality. A_2 was accentuated and greater than P_2. The blood pressure was 100/85. The abdomen was soft although there was some tenderness in the right upper quadrant. The liver and spleen were not palpated. The remainder of the physical examination was negative.

The temperature was 100.2°, the pulse 100. The respirations were 28.

Examination of the urine showed a specific gravity of 1.010 to 1.018 with a large trace of albumin. The sediment contained an occasional white blood cell and a rare granular cast, but was otherwise negative. The blood showed a red cell count of 4,900,000, with a hemoglobin of 70 per cent. The leukocytes numbered 16,500,

with 80 per cent polymorphonuclears, 2 per cent eosinophils, 2 per cent basophils, and 16 per cent lymphocytes. Blood cultures were negative. The nonprotein nitrogen of the blood was 50 milligrams per cent. The sputum contained pneumococcus, not types I, II or III. An electrocardiogram exhibited a prominent P_1 and P_2. There was high ST takeoff with a humping of T_1 and a very low T_2. Q-R-S_3 was inverted. Lead 4 exhibited an absent Q deflection and ST_4 took off from the descending limb of R, 3 millimeters below the isoelectric level. T_4 was inverted.

The pain and dyspnea persisted with very little change despite the administration of morphin. A pleuropericardial friction rub was reported to be audible over the third right interspace close to the sternal border. This, however, was not confirmed by other examiners. The râles became more pronounced at both bases and pitting edema appeared at the sacrum. On the eighth day the electrocardiogram revealed a slight left axis deviation with a prominent S_2. ST_1 was convex with a slightly high origin and inversion of T_1. T_2 and T_3 were likewise inverted. Lead 4 exhibited an absent Q, a tall R and a low origin of S-T with a diphasic T. Throughout the hospital stay his temperature remained normal, the pulse ranged from 80 to 90 and the respirations were 20. His condition remained essentially unchanged until the fourteenth day when he developed very severe precordial pain and dyspnea while his bed was being made and expired within fifteen minutes.

Differential Diagnosis

Dr. Paul D. White: "During this time he also had knife-like pains in the small of his back and stated that there was swelling of all parts of his body which persisted for about six weeks." One would like a report of the physical examination at that time. It is evident that we cannot be sure of the diagnosis of this attack without further information. There are a number of things we should think of: congestion or heart failure associated with or due secondarily to luetic aortitis with aortic valvular disease or coronary mouth involvement; chronic hypertension with a hypertensive heart which had failed at this time; coronary thrombosis with failure ensuing; chronic valvular disease, perhaps with aortic stenosis which is often found in middle and in old age at the beginning of heart failure; acute pericarditis with effusion; and even pulmonary embolism, although we would not expect edema with that. Also, if the edema was widespread we should have to think of the possibility of uremic involvement.

The edema subsided. We would like to know what treatment he had had during this time because if he had had very little treatment in the way of digitalis and rest we could rule out most of the conditions mentioned above. An acute attack of pericarditis or an acute attack of coronary thrombosis may result in symptoms and signs that will subside spontaneously without much treatment. You would hardly expect luetic aortitis severe enough to cause failure to subside in this way. Coronary thrombosis is the best bet.

Evidently we have hypertension on recovery from the acute illness and an enlarged heart with a systolic murmur at the apex. What are the causes of such a murmur at the apex? The best and most likely explanation is that the heart is still somewhat dilated and that the murmur is a functional murmur due to enlargement of the left heart. Second in likelihood would be aortic stenosis with the murmur heard best at the apex. A number of times in the past ten years we have been misled by such a murmur, later discovering that the murmur became louder at the aortic valve area. The least likely possibility is mitral disease causing this systolic mitral murmur. The fact that the liver edge was felt is important and favors an acute illness complicating hypertensive heart disease. An acute illness that would explain the precordial constriction followed by this wheezy respiration would be coronary thrombosis, so that the history to this point somewhat favors the combination of hypertensive heart disease with acute coronary occlusion. Hypertensive heart disease itself may be complicated by failure, but we would not expect precordial constriction or pain to be a prominent feature if that were so.

We cannot apparently blame the albuminuria on congestive failure. It does seem as though there must have been some degree of nephritis, quite likely secondary to the hypertension.

The Hinton reaction is very important in a middle-aged man with heart symptoms. In such a patient we must think of the possibility of luetic aortitis which may show itself by the symptom of oppression under the sternum or by dyspnea. This negative Hinton test is of value; it is against the diagnosis of syphilitic aortitis by about four to one.

The heart was probably slightly enlarged but not dilated at this time, or if it was dilated, not enough so to cause abnormal measurements by x-ray examination; but there is a suggestion in the x-ray report that the apex was blunted and the aorta tortuous that would be in keeping with aortic stenosis or with hypertension, plus aortic sclerosis.

It would be of interest to know if an electrocardiogram was obtained on the first admission.

"Systolic and diastolic murmurs were audible at the left sternal border." This is the only statement about the diastolic murmur. I would

like to know if the diastolic murmur was heard constantly, and if anybody heard it at the time of the first admission; there is no note later on that it was present or that it was only a temporary finding.

All these symptoms can be explained by progressive congestive heart failure.

"Three weeks before admission he began to suffer from a 'tight pain' behind the middle of the sternum following the slightest effort." That is angina pectoris certainly. I wonder if at the first illness two years before he might have had the same story. The story, of course, makes one think of acute block of the coronary vessel due to infarction. The prolonged pain was similar in character to the angina pectoris pain.

It hardly seems as if he had congestive failure enough to cause him to raise bloody sputum, but that is a possibility. One should also think of pulmonary infarction with pleurisy complicating the coronary thrombosis as a possible explanation for the pain in the right chest, the cough and the raising of blood. The findings on physical examination support our opinion that there was some involvement of the right lower lobe, probably infarction.

There is no statement of murmurs found in the physical examination at the second admission; we would very much like to know about the aortic diastolic murmur that he had had.

"The blood pressure was 100/85." That is a striking drop in blood pressure and would be consistent with coronary thrombosis or with any other condition in which there was an acute heart or circulatory failure.

There is some importance in the statement that the spleen was not palpated. No note is made of the clubbing of the fingers. Later on blood cultures were taken. Apparently there was a thought of bacterial endocarditis.

The classical leads of the electrocardiogram were not very striking at first although there was stated to be a high origin of T_1, low T_2, and rather low origin of T in lead 3. From lead 4, the precordial lead, however, we received much more help. The so-called Q wave which ordinarily is well marked was absent; so far as we know that is fair evidence of an established infarct, not necessarily an acute infarct, in the left ventricle. Also the T arose from the descending limb of the R wave, very low down. This change was probably associated with the acute illness.

"A pleuropericardial friction rub was reported to be audible over the third right interspace close to the sternal border." That was due to pleurisy associated with the pulmonary infarct or to pericarditis secondary to the coronary thrombosis, if such was present.

In the second electrocardiogram the three classical leads present much more evidence than we had before of what seems to be an acute process. The T wave in lead 1 has become inverted, which is an important change, and the T in lead 2 has become inverted, which is also important. Thus, T_1 and T_2 are now definitely inverted, while in lead 4 the Q is still absent and the T rises farther up on the descending limb of the R.

We cannot diagnose with certainty secondary coronary occlusion, or rupture, or angina pectoris; the last-mentioned possibility is least likely because he lived fifteen minutes after the pain began. There are many remote possibilities as to the exact cause of death.

I believe that the diagnosis here is hypertensive coronary heart disease with an old myocardial infarct and probably a fresh one, a complicating pulmonary infarction, and congestive failure, the heart being unable to stand the extra strain. I do not think that luetic aortitis is present; it cannot explain the whole picture, particularly the electrocardiogram, as it is given. We have no proof of valvular disease. Aortic stenosis and regurgitation would have to be thought of and possibly careful repeated examinations might have confirmed those murmurs heard at first, but the chances are against such a diagnosis. From the evidence we can have a hypertensive complication of aortic stenosis, but that is not likely either. Remote possibilities of dissecting aneurysm, bacterial endocarditis, and such a rarity as dissection of the coronary artery, I think we can rule out.

Dr. Gerald Blake: This patient's symptoms of angina recurred about three weeks before his second admission and then only following exertion. Four days before admission he had what we thought was coronary occlusion and he came in with the picture of coronary occlusion with signs of myocardial failure. He described his pain as burning during the first day or two, "pain like fire", and, following that, as a stabbing pain at the root of the sternum, going through to the back. The signs in the lungs were as described and could be explained as chronic passive congestion, with probably an infarction on the right. His final attack was accompanied by a slight increase in dyspnea, slight increase in cyanosis and slightly more rapid breathing, and we thought that that was probably due to occlusion of the main left coronary vessel rather than to a rupture of the myocardium.

Dr. Howard Sprague: I saw the patient once in consultation on the ward. I agreed at that time that the diagnosis was the one that Dr. White has outlined. I did hear at that time what seemed to be a pericardial friction rub. The electrocardiogram certainly suggests the apical type of coronary occlusion.

Clinical Diagnoses

Coronary thrombosis.
Generalized arteriosclerosis.

Chronic vascular nephritis.
Pulmonary infarct.

DR. PAUL D. WHITE'S DIAGNOSES

Hypertensive, coronary heart disease.
Coronary thrombosis (anterior descending branch of the left coronary artery).
Infarcts of apex of left ventricle, old and new.
Pulmonary infarct.
Congestive failure.
Angina pectoris (history).

ANATOMIC DIAGNOSES

Coronary thrombosis, right descending branch.
Myocardial infarction.
Pericarditis, acute fibrinous.
Mural thrombi, left ventricle.
Pulmonary edema, bilateral.
Hydrothorax, right.
Pulmonary atelectasis, right.
Arteriosclerosis: Coronary and cerebral, marked; aortic, moderate.
Perihepatitis.
Perisplenitis.
Chronic vascular nephritis.
Oxycephaly.
Obesity.
Parathyroid hyperplasia, slight.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The autopsy showed a large area of infarction in the heart at the apex of the left ventricle involving nearly two-thirds of the interventricular septum. The area of infarction was soft, the muscle entirely different in color from the remainder of the myocardium. It was evident that it had been present long enough to get a considerable degree of reaction but not long enough for complete scarring. From the myocardial infarct itself we were not able to make out any clear evidence of successive infarctions, though the degree of muscle cell degeneration varied slightly from place to place. There were, however, two quite separate thrombi overlying the infarct on the endocardium of the left ventricle. These were evidently of different age, one was firmly and almost completely organized, the other showed only the very slightest trace of organization so I feel pretty sure we are dealing with two successive infarcts occupying almost exactly the same spot in the myocardium. The coronary that was involved was the descending branch of the right. Peculiarly enough, the left showed marked sclerosis but was not occluded.

DR. WHITE: The area involved was the apex of the left ventricle?

DR. MALLORY: Yes. One feature that impressed me particularly at the autopsy was the extent of the pericarditis. The entire pericardial cavity was involved in a very severe fibrinous pericarditis showing early signs of organization.

DR. WHITE: I meant to have said that a pericarditis could account for some modification of the electrocardiogram but not to the extent found here.

DR. MALLORY: The average case of coronary thrombosis will almost invariably show localized pericarditis over the area of infarction, but diffuse pericarditis such as this is really quite unusual and I think it might explain the very marked radiation of pain through to the back.

A PHYSICIAN: Was the pleura involved too?

DR. MALLORY: The right pleural cavity contained about half a liter of pleural effusion and there was collapse of the lower lobe on the right, no pulmonary emboli, no infarcts.

The other organs showed grossly only marked arteriosclerosis, particularly of the abdominal aorta and of the vessels of the circle of Willis. Microscopically there is an early arteriolarsclerosis such as one would expect in hypertension showing up most markedly in the kidneys.

DR. WHITE: May I add one note about the electrocardiogram evidence? The classical leads, that is, leads 1, 2, and 3, were not wholly convincing in the first record, but the fourth or chest (precordial) lead gave a great deal of help through the absence of the so-called Q wave. This is an illustration of the occasional value of the routine chest lead.

CASE 22132

PRESENTATION OF CASE

A thirty-six year old white Portuguese rayon mill worker was admitted complaining of epigastric discomfort and loss of weight.

About a year and a half prior to entry several hours after his evening meal the patient felt dizzy and shortly afterward had a desire to defecate. He passed a very black stool and fainted. Upon recovering consciousness after four or five minutes he found himself lying in a pool of dark blood which he believed he had vomited. He was sent to a hospital where x-ray studies were said to be negative. A Sippy diet was instituted and the patient was discharged much improved after three to four weeks. He felt well for about six months, whereupon he observed some pallor, weakness, and slight dyspnea with exertion. Suddenly one evening he had a sensation of fullness in the chest and expectorated a cupful of dark blood which was streaked with red. For two succeeding days there again were tarry stools. The Sippy régime was reinstituted and he continued well except for a sensation of epigastric fullness which occurred after meals. This was relieved by eructation. About ten months before admission he began to have a tight gripping sensa-

tion in the midepigastrium. This usually occurred immediately after breakfast and persisted throughout the day with an exacerbation of intensity following each meal. Relief was spontaneous after rest at night but medication had no effect. A milk and cream diet resulted in relief from this symptom after about two months. At this time a new symptom manifested itself: all ingested food appeared to stop momentarily behind the xiphoid, cause a slight pain and a feeling of fullness, pass with a rather sharp pain, and leave a residuum of burning pain for about ten to fifteen minutes. Dry solid food, such as toast, caused the discomfort to persist for the entire day. Another x-ray at this time was negative. During the succeeding months he became progressively weaker, short of breath, and his weight decreased from about 130 pounds to 105 pounds. There were no further tarry stools. He continued to work up to three weeks before his entrance to this hospital. The skin of his face and forearms had become slightly browned during the last two years. This he attributed to the action of sulphuric acid fumes with which he had considerable contact.

Physical examination showed a poorly nourished white male in no acute distress. An acneform eruption was noted over the upper arms and back. The mouth exhibited extensive pyorrhea with brownish-blue pigmentation of the upper and lower gums. The pharynx was injected and there were three small hemorrhagic spots upon the mucosa of the hard palate. The lungs were clear and the heart normal. The blood pressure was 94/78. The liver extended four fingerbreadths beneath the costal margin. It was not tender but felt hard and nodular. Rectal examination elicited only a single small thrombosed hemorrhoid.

The temperature, pulse, and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 5,080,000, with a hemoglobin of 90 per cent. The white cell count was 10,000, 69 per cent polymorphonuclears. A Hinton test was negative.

A gastrointestinal series showed a circular mass lying on the left side of the lower end of the esophagus forming a lobulated margin. There was rigidity of the upper third of the lesser curvature including the fundus with bulging in this region. The entire stomach was slightly displaced to the left and there were pressure defects in the lower end of the antrum, on the cap and the upper flexure of the duodenum.

One week after entry the patient suddenly complained of severe upper abdominal pain which was most severe on the left side. The pain rapidly became generalized and the abdomen became rigid, tender, and a tympanitic note was obtained by percussion over the liver area. An x-ray of the abdomen showed free air beneath both leaves of the diaphragm. A laparotomy was performed.

Differential Diagnosis

Dr. Arthur W. Allen: Will you demonstrate the x-rays, Dr. Schatzki?

Dr. Richard Schatzki: Here is the area where a temporary stop in the barium column was described about two inches above the diaphragm. This is the outline of the esophagus, the left margin is irregular, whereas the right side is fairly regular. This irregularity continues down to here, involving the upper third of the stomach. This region was rigid. There was displacement of the stomach to the left. It was very difficult to fill the cap and second portion of the duodenum, apparently due to pressure from outside. On this spot film the defect in the lower end of the esophagus is better demonstrated.

Dr. Allen: This case is that of a young man whose first symptom is that of severe gastrointestinal hemorrhage. The fact that he had no great pain at the time of the onset of the hemorrhage would not be particularly noticeable if you were going to try to explain the severe bleeding from the most common cause, that is, duodenal ulcer. A certain number of duodenal ulcer patients do not have any pain before they have a rather severe hemorrhage. The fact that his x-rays were negative after this massive hemorrhage would not necessarily rule out a bleeding duodenal ulcer as a cause, because these are usually on the posterior wall of the duodenum and it takes a rather special technique to demonstrate a lesion in this region. Or if the lesion which caused his hemorrhage was at the cardia or high on the lesser curvature of the stomach, near the esophagus, the x-ray examination might be negative. It is, I believe, a difficult place to show by some of the ordinary routine gastrointestinal methods.

The fact that the Sippy diet relieved him of his difficulty for a while is not particularly significant because if he had a lesion in his lesser curvature or in the duodenum I think he might have been relieved by such a diet; any bland diet would have helped him temporarily, particularly with the rest that went with it. The new symptom of discomfort after he ingested food, which developed about eight months prior to admission, is strongly suggestive of some actual narrowing of the opening of the esophagus into the stomach.

He comes in with loss of weight, with a brown skin. One wonders how much you can tell about the color of the skin in a man of this nationality because a great many Portuguese are dark. I am sure I have no idea what possible bearing the sulphuric acid fumes could have on this case. He does have very low blood pressure of

94/78 which with his brown pigmented skin might suggest Addison's disease or something of that nature, but I believe in emaciated patients, particularly with malignancy, we also see, in the terminal stages, a type of skin with lowered blood pressure, and so forth, that is not unlike this.

The fact that his red blood cell count was normal is a little disturbing. One would expect a man with this amount of loss of weight, with the amount of difficulty he had had ingesting proper food and with a lesion that is as large as this involving the lower end of the esophagus and stomach, to show a certain amount of anemia. It is a little confusing that we have such a perfect blood picture.

Now as to the various causes of severe gastrointestinal hemorrhage, the commonest is duodenal ulcer and after that I presume we must put gastric ulcer, then an esophageal varix, then polyp of the stomach, and lastly carcinoma. These are the things that can and do commonly account for massive hemorrhage from the upper gastrointestinal tract. Now can this man have had a bleeding duodenal ulcer in the beginning, and an associated lesion high in the stomach or lower end of the esophagus, and could hemorrhage come from the duodenal ulcer and his symptoms be relieved by Sippy diet? We do not get any help from the type of bleeding which he had as to where it came from particularly, because in the duodenal ulcer cases about one-third vomit the blood, about one-third pass it entirely by rectum and the other third pass the blood in both directions, which is what this man did.

The final admission with exitus occurring as it did would mean he had a perforation of a hollow viscus, most likely his stomach or the lower end of the esophagus, producing gas under the diaphragm. With the history as it is given there can be no doubt but that he had a perforated hollow viscus and it is most likely in this neighborhood. Operation was performed which I would anticipate as being unsuccessful, because if a lesion of this size and character perforates one could never hope to close such a perforation surgically. Perforation of a malignant lesion of a hollow viscus is usually fatal. It almost invariably is fatal regardless of what portion of the gastrointestinal tract it involves. It is not always fatal in the colon but it is very apt to be.

I should suppose that we would have to be prepared to find that this man had an old scar in the duodenum which might have accounted for his first hemorrhage. On the other hand I am inclined to try to explain it all on the basis of this lesion at the lower end of the esophagus and the upper end of the stomach and I believe that he had a malignant lesion here possibly developing upon ulcer which finally perforated, causing his death.

Dr. Rackemann has called my attention to the big liver. I should have mentioned that. I suppose a large, hard, nontender, nodular liver is perfectly consistent with a malignant growth of this size and we might expect to find that the liver was full of metastatic cancer.

Clinical Diagnoses

Carcinoma of the stomach with metastases.
Bronchopneumonia.
Perforation of the stomach.

Dr. Arthur W. Allen's Diagnoses

Carcinoma of the lesser curvature of the stomach and the lower end of the esophagus.
Perforation of the malignant growth.
Metastasis to the liver.
Peritonitis.

Anatomic Diagnoses

Carcinoma of the stomach with perforation and metastases to the liver, regional lymph nodes and pelvis.
Bronchopneumonia, bilateral.
Pulmonary edema and congestion, bilateral.
Pleuritis, chronic fibrous, bilateral; acute fibrinous, left.
Hydrothorax, left, slight.
Peritonitis, acute fibrinopurulent, and chronic fibrous.
Subphrenic abscess, left.
Perisplenitis.
Arteriosclerosis, slight.
Emaciation, marked.

Pathologic Discussion

Dr. Tracy B. Mallory: The autopsy showed a carcinoma which involved the last two and a half centimeters of the esophagus and the upper three and a half centimeters of the stomach. Whether it was primary in the base of the esophagus or in the cardia of the stomach is pretty difficult to say. I, personally, am inclined to think it was primary in the stomach. It had perforated of course. We found the perforation on the anterior surface of the stomach close to the cardiac orifice but only with some difficulty since it proved to be not much over a millimeter in diameter.

The case interested us a good deal from the character of the metastases. You will remember that a few months ago we presented a case of cancer of the esophagus in which nodules of metastatic cancer ran upward from the primary growth, a considerable distance up the esophagus, so that an x-ray characteristic of esophageal varices was produced. This man showed the same thing to a somewhat less degree.

There was a whole string of submucosal nodules running up the esophagus. Another point of some interest was that the metastases to the liver were all of them in the left lobe. The liver was adherent to the growth and it is possible that it was involved by direct extension. The other possibility is that it is to be explained on Graham's theory, that blood from certain parts of the portal system tends regularly to go to the left lobe while that from other areas goes to the right lobe. The cardia of the stomach would be in the area which tended to drain into the left lobe.

There was a well-marked subdiaphragmatic abscess on the left, a little reactive irritative pleuritis in the right pleural cavity and a terminal bilateral pneumonia.

A PHYSICIAN: Was there any effusion in the chest?

DR. MALLORY: A few cubic centimeters of turbid fluid.

A PHYSICIAN: Was there anything in the adrenals?

DR. MALLORY: No.

A PHYSICIAN: Was the perforation successfully closed at operation?

DR. MALLORY: No.

A PHYSICIAN: And you found the diaphragm normal?

DR. MALLORY: Yes.

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JOHN SCOTT HALDANE

THE death of John Scott Haldane on March 15, 1936, removes a great international figure in physiology and scientific medicine—a personality as remarkable in some respects as Professor Pavlov whose death was announced three weeks ago in these columns. J. S. Haldane came of the Haldanes of Gleneagles, a family long distinguished for its brilliant intellectual attainments. His father, Robert Haldane, was a well-known Scottish barrister (writer to the *Signet*), his mother, Mary Burdon Sanderson, was a sister of John Burdon Sanderson, the first incumbent of the chair of physiology at Oxford; his sister, Elizabeth Sanderson Haldane, a woman of high attainment in philosophy and letters, is a foremost authority on Descartes; his brother, Viscount Haldane (Richard Burton Haldane, 1856-1928) was the well-known British statesman and War Minister; and finally his son, Professor J. B. S. Haldane, and daughter, Naomi Mitchison, also exemplify the versatility and remarkable talent of their line.

After his preliminary education at the Edinburgh Academy, J. S. Haldane went to Oxford where he received training in physiology under his uncle, Burdon Sanderson; after a short period in London, during which time he received his medical qualifications, he returned to Oxford as Demonstrator in Physiology (being later appointed Reader), and he remained at Oxford until the end of his life. The facilities of the Oxford Laboratory were so meager that Haldane erected a private laboratory in his house, and during the last twenty-five years most of his investigations were carried out under his own roof, and unfortunately students seldom had contact with him.

Haldane will always be remembered for his pioneer studies upon the physiology of respiration; he introduced a simple method for analyzing the constituents of expired air, his apparatus for gas analysis now being found in every physiological laboratory in the world. Haldane's most celebrated paper was published with J. G. Priestley in 1905. It bore the title, "Regulation of the lung-ventilation" (*Journal of Physiology*, **32**, 225-266, 1905). In an ingenious series of experiments carried out upon themselves they proved that:

> "The respiration centre is exquisitely sensitive to any rise in alveolar CO_2 pressure; a rise of 0.2 per cent of an atmosphere in the alveolar CO_2 pressure being for instance sufficient to double the amount of alveolar ventilation during rest. (See table.)
>
> "When the oxygen pressure in the expired air falls below about 13 per cent of an atmosphere, the respiratory centre begins to be excited by want of oxygen and the alveolar CO_2 pressure begins to fall."

This is a classical study in the history of physiology, a landmark not only in the field of respiration, but in the larger field of clinical science, since it illustrated in a beautiful manner how well-controlled physiological studies might be carried out on human beings. Haldane's abiding interest in respiration continued until the end of his life. His Silliman Lectures given at Yale in 1916 culminated in a monograph bearing the title *Respiration*, originally published by the Yale Press in 1922, and just reprinted after complete revision with J. G. Priestley in 1935. It would be impossible to comment fully on the importance of this book; many felt that it had rather glaring shortcomings, which arose out of Haldane's philosophical turn of mind. In the first edition the chapter on oxygen secretion by the lungs caused some who had done far less work than Haldane to shrug their shoulders; and while there is room for disagreement concerning this phase of Haldane's work, there are few scientific expositions which have so sharpened the critical faculties of students, and

the book itself remains as the only comprehensive monograph in English on the physiology of respiration.

	Subject and number of experiment	% of CO_2 in inspired air	Average depth of respirations in cc.	Average frequency of respiration per minute	Depth of respirations, normal=100	Frequency of respirations, normal=100	Volume breathed per minute, normal=100	Ventilation of alveoli with inspired air, normal=100	Calculated CO_2 % in alveolar air
J. S. H.	2	0.79	739	14	112	100	111	116	5.5
"	2	1.47	978	13	147	93	137	149	5.0
"	2	1.52	793.5	15	120	107	128	137	5.55
"	1	1.97	849	13.5	150	84	128	147	5.7
"	2	2.02	864	15	130	107	139	153	5.6
"	4	2.28	911	15	142	100	141	161	5.8
"	3	2.31	919	14.5	155	91	140	164	5.7
"	4	2.84	1154	16	178	107	191	227	5.3
"	4	3.07	1216	15	189	100	186	226	5.5
"	4	3.11	1232	15	192	100	191	230	5.5
"	3	3.73	1330	14	224	88	196	273	5.9
"	3	4.84	1662	14	284	88	245	322	6.5
"	1	5.14	1771	19	313	120	373	498	6.2
"	3	5.48	1845	16	311	100	311	411	6.8
"	1	6.02	2104	27	372	169	631	857	6.6
J. G. P.	5	0.60	516	15	103	100	103	104	6.6
"	2	1.64	605	19	116	112	130	136	6.2
"	1	1.65	540	15.5	121	94	114	123	6.7
"	5	2.53	664	16	133	107	141	155	6.5
"	6	2.62	747	15	150	94	140	158	6.6
"	2	2.63	750	18	144	106	152	170	6.3
"	1	2.71	570	17	128	103	132	144	7.0
"	6	3.22	922	15	185	94	168	203	6.3
"	2	3.23	750	17	144	100	144	160	7.1
"	5	3.42	791	16	159	107	168	192	6.6
"	3	3.47	465	17.5	144	107	154	189	6.8
"	1	3.51	724	20	162	121	197	231	6.2
"	4	3.89	698	18	168	106	178	215	6.8
"	2	4.16	935	18	179	106	190	218	7.0
"	1	4.16	895	21	201	127	255	314	6.1
"	4	4.56	969	19.5	233	114	267	343	6.3
"	3	4.58	575	20.5	177	126	223	297	6.6
"	3	4.88	760	20	234	123	288	413	6.35
"	3	5.22	910	21	281	129	362	540	6.3
"	4	5.23	1094	19.5	263	114	301	395	6.8
"	4	5.67	1331	20.5	320	121	385	520	6.8

Haldane and Priestley's original table showing the relation between CO_2 tension in alveolar air and lung ventilation.

Haldane rendered great service to his country and to applied physiology generally through his studies of respiratory hazards: e.g., in mines, tunnels, caissons, and various other conditions of altered atmospheric pressure or contamination of the atmospheric air. The distinguished work of Henderson and Haggard in this country was largely influenced by Haldane's pioneer studies as was that of many physiologists on the continent.

Philosophically Haldane has generally been referred to as a vitalist. He was exceedingly impatient with the naïve postulates of behaviorism, tenaciously maintaining that living matter could never be accounted for by physics and chemistry alone. His many efforts to harmonize philosophy, science and religion will be found in: *Essays and philosophical criticisms,* 1883; *Mechanism of life and personality,* 1913; *Organism and environment,* 1917; *The New physiology,* 1919; *The sciences and philosophy* (Gifford Lectures), 1929; *The philosophical basis of biology,* 1931; *Materialism,* 1932; *The philosophy of a biologist,* 1935.

Personally Haldane was a man of unusual charm. For some years he had become progressively stooped and had to cock his head in a curious manner in order to make his face visible to an audience, but when his eye caught yours it seemed to penetrate your soul. In manner he was detached and, as with Burdon Sanderson, the stories are legion of his absent-minded ways. Students of Oxford saw him more frequently catching a train a second or two before it was due to depart than they did in his laboratory; but those who were privileged to see him in his house found him a gracious and most fascinating host.

"THE OLD DOCTOR'S ALMANAC"

CLIMATOLOGY has long been suspected of having its alliance with medicine, and climate its influence upon disease; rickets is less florid in the sunny South than in our fog-bound northern climes, and is practically unknown among the rank verdure of the tropics; rheumatic fever hides its face in sunny latitudes and sufferers from asthma find relief in the dry highlands of Arizona.

It has remained for an Austrian physician, however—a member of the Vienna Board of Health, according to our esteemed contemporary, the *New York Times*—to elaborate a calendar of diseases corresponding to the alternations of climate with variations of temperature, humidity and barometric pressure. This Almanack, recently published, represents a labor of eight years and a study of some 20,000 cases.

According to this calendar, "January is the month of measles; February brings snowdrops and hay fever; March is the month of pneumonia; the appearance of the lily of the valley in May corresponds with the increase in asthma and appendicitis.

"June roses herald the troubles connected with the gallbladder and liver; July increases the sufferers from heart trouble; August is the month of unclassified malaises; September sees the beginning of the 'cold in the head' season, which reaches its height in November.

"December is the month of digestive troubles, which are largely caused by the lack of fresh vegetables."

Perhaps with this almanac, synoptically printed, a vade mecum may be available which will do away with many a bulky textbook of disease, and a new era of more precise diagnosis will be inaugurated. Who knows but that, given a few years of trial—and years are fleeting—and the sling psychrometer and the aneroid barometer will replace the stethoscope and the sphygmomanometer, and the relief of abdominal distention will be measured in terms of wind velocity?

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

HARMER, TORR WAGNER. A.B., M.D. Harvard University Medical School 1907. F.A.C.S. Instructor in Anatomy, Harvard University Medical School. Assistant Visiting Surgeon, Massachusetts General Hospital. Consulting Surgeon, Arlington-Symmes Hospital; Massachusetts Ear and Eye Infirmary; Somerville, Waltham and Winchester Hospitals. His subject is Certain Aspects of Hand Surgery. Page 613. Address: 416 Marlborough Street, Boston, Mass.

MUNRO, DONALD. A.B., M.D. Harvard University Medical School 1916. F.A.C.S. Visiting Surgeon in Charge of Neurological Surgery, Boston City Hospital. Assistant Professor of Neurological Surgery, Harvard University Medical School. His subject is The Activity of the Urinary Bladder as Measured by a New and Inexpensive Cystometer. Page 617. Address: Boston City Hospital, Boston, Mass.

MOCK, HARRY E. B.S., D.Sc., M.D. Rush Medical College 1906. F.A.C.S. Senior Attending Surgeon, St. Luke's Hospital. Associate Professor of Surgery, Northwestern University Medical School. His subject is Management of Skull Fractures. How Can the High Mortality Rate Be Reduced? Page 625. Address: 122 South Michigan Boulevard, Chicago, Illinois.

IRVING, FREDERICK C. A.B., M.D. Harvard University Medical School 1910. F.A.C.S. William Lambert Richardson Professor of Obstetrics, Harvard University Medical School. Visiting Obstetrician, Boston Lying-in Hospital. Consulting Obstetrician, Newton Hospital. His subject is The Mechanics of Delivery Especially as it Relates to Intracranial Hemorrhage. Page 635. Address: 221 Longwood Avenue, Boston, Mass.

KELLOGG, FOSTER S. A.B., M.D. Harvard University Medical School 1910. Associate in Obstetrics, Harvard University Medical School. Assistant Visiting Obstetrician, Boston Lying-in Hospital. His subject is The Prevention of Puerperal Infection. Page 636. Address: 19 Bay State Road, Boston, Mass.

BEDELL, ARTHUR J. M.D. Albany Medical College 1901. F.A.C.S. Attending Ophthalmologist, St. Peter's and Child's Hospitals, and Old Ladies Home. Consulting Ophthalmologist, Anthony N. Brady Maternity Hospital, Albany; Physicians Hospital, Plattsburg; Vassar Brothers Hospital, Poughkeepsie; Little Falls Hospital, Little Falls; and Moses Ludington Hospital, Ticonderoga. Fellow of the American Ophthalmological Society and of the Academy of Ophthalmology and Oto-Laryngology. His subject is The Causes of Sudden Blindness. Page 640. Address: 344 State Street, Albany, New York.

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DYSMENORRHEA

There are two types of dysmenorrhea, primary and secondary. Primary dysmenorrhea is uncomplicated by any pelvic pathology capable of causing uterine pain. Secondary dysmenorrhea is due to accompanying inflammations, tumors, etc. It is with primary dysmenorrhea that we are concerned. The symptoms and diagnosis need no elaboration, for the term itself is sufficiently descriptive.

The treatment of primary dysmenorrhea is difficult and often disappointing but, if general rules of procedure are laid down and followed, the patient will obtain relief in the end. The outline should proceed from the simplest method to the most difficult or serious. All possibilities of a psychic cause for dysmenorrhea must be ruled out before any other treatment is instituted. The anxious mother or older sister should be eliminated.

Psychiatric treatment is successful infrequently but it must be tried if there is any suspicion, for other methods will fail if the main cause is psychogenic.

Primary dysmenorrhea may in some instances be assumed to be of endocrine etiology and if this is true some hormone should be of aid in the treatment. Many patients with dysmenorrhea have an accompanying underdevelopment of the uterus and to develop such a uterus should be the aim of the physician. In experimental animals and in some instances in human beings the use of estrogenic substances has proved successful in enlarging the underdeveloped uterus. The use of estrin is therefore justifiable. It is also true that estrin causes a marked sensitization of the uterine smooth musculature to the posterior pituitary hormone pituitrin (oxytocic principle). The corpus luteum hormone progestin is known to neutralize estrin and thus desensitize or prevent the sensitization of the uterine musculature. Progestin can therefore be used with some hope of success in patients with an oversensitive uterine

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

musculature. Urinary prolan (luteinizing factor) or pituitary prolan (luteinizing factor) can be utilized in the same way. The hormone should cause luteinization of the follicles and thus the patient's own progestin will neutralize the sensitizing estrin. It is known that estrin will inhibit the pituitary from secreting prolan. If therefore there is an excess of estrin, estrin can be given the patient with the hope of preventing further secretion of prolan and therefore less secretion of the patient's own estrin. The estrin should be omitted a few days before the onset of the menstrual period and it is assumed that in the time left between omitting estrin and menstruation the patient's prolan will not have had time to cause enough estrin secretion to sensitize the uterine musculature. The above are the theories for the use of hormones in dysmenorrhea.

If endocrine methods of attack fail, the simplest surgical procedures should be tried. Sometimes it is noticed that, after taking an endometrial biopsy in the office, the next menstrual period is less painful. In taking an endometrial biopsy there may be a slight stretching of the internal os and it can be assumed that this procedure relieved the pain. It seems logical then to try further dilatation in the office. By infiltrating the os with novocain or using cotton pledgets of local anesthetics (nupercaine, pontocaine) in the os or by using evipal in small doses moderate dilatation can be done without much discomfort. If the dilator is well lubricated it is surprising how easily a fairly satisfactory dilatation can be accomplished.

When this type of treatment has proved unsatisfactory the external os should be thoroughly dilated under an anesthetic. This dilatation should be careful, slow, and very thorough. It is not sufficient simply to pass the dilators, but a real dilatation must be kept up for at least fifteen minutes. Often when doing the rectal examination a widening of the uterocervical segment is noted. This thickening is similar to the hard area noted about the pylorus in pyloric obstruction in children. It is sometimes difficult properly to dilate this area and it is safe and proper to section it. With a finger in the rectum and a long pointed knife this area can be cut through longitudinally and a piece of gauze packed in the split area to keep it open. After regular dilatation and dilatation with incision the internal os should be kept open by dilatation with a lubricated dilator in the office. This method insures a longer and more satisfactory result from surgical dilatation.

Some dysmenorrheas cannot be relieved by these methods and if, in a virgin, a retroflexion is found or if in a multipara a large boggy retroverted uterus is found, suspension of such a uterus combined with dilatation of the internal os is justifiable. Suspension of the retroverted virginal uterus or of the anteflexed virginal uterus is not so frequently followed by relief of pain.

Lately presacral or superior hypogastric neurectomy for relief of pain has been advocated. Without doubt this method of treatment is eminently satisfactory. If at any abdominal operation sufficient reason for the dysmenorrhea is not found it should be performed. No ill effects to the bladder, bowel, libido, or pregnancies have been reported. It will not cure all patients. In some the pain which seems organic will be found to be psychogenic and no relief obtained from the operation. In others aberrant nerves or lack of sufficient radical excision of the nerves will not relieve the pain as expected. It is not a 100 per cent cure but is successful in about 80-90 per cent of cases.

Last of all if the pain is intractable and no method can relieve it hysterectomy is justifiable. This is especially true in older women who have had children and in older virgins who understand what hysterectomy means to them. In youth it should never be necessary and should be reserved for later life.

THIRD ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning March 29:

Berkshire

Thursday, April 2, at 4:30 P.M., at the House of Mercy Hospital, Pittsfield. Subject: Dermatology—Ten Common Skin Diseases—Diagnosis and Treatment, (1) Impetigo Contagiosa, (2) Scabies, (3) Acne Vulgaris, (4) Psoriasis and Seborrhoeic Dermatitis, (5) Epidermophytosis, (6) Herpes Simplex and Zoster, (7) Eczema, (8) Erythema Multiforme, (9) Verruca Vulgaris and (10) Dermatitis Medicamentosa and Dermatitis Venenata. Instructor: J. H. Swartz. Melvin H. Walker, Jr., Chairman.

Bristol North

Wednesday, April 1, at 7:30 P.M., at the Morton Hospital, Taunton. Subject: Kidney and Bladder Diseases A and B (Medical)—Acute and Chronic Nephritis. Instructor: E. M. Chapman. Arthur R. Crandell, Chairman.

Bristol South (New Bedford Section)

Friday, April 3, at 4:00 P.M., at the St. Luke's Hospital, New Bedford. Subject: Kidney and Bladder Diseases A and B (Surgical)—Hematuria; Its Significance in Surgical Diseases of Kidney and Bladder. Prostatism and Related Diseases. Cystitis and Pyelitis. Instructor: G. G. Smith. Harold E. Perry, Chairman.

Norfolk*

Friday, April 3, at 8:30 P.M., at the Norwood Hospital, Norwood. Subject: Arthritis—(a) Medical Care of Patient in the Home. (b) Orthopedic Treatment in Hospital and Aids in Home Treatment. Instructors: A. A. Hornor and J. S. Barr. H. B. C. Riemer, Chairman.

Worcester (Milford Section)

Wednesday, April 1, at 8:30 P.M., at the Milford Hospital, Milford. Subject: Kidney and Bladder Diseases A (Medical)—Acute Nephritis — Etiology, Diagnosis and Treatment. Nephrosis and Its Treatment. Instructor: L. B. Ellis. Joseph I. Ashkins, Sub-Chairman.

*The Course previously given at the Faulkner Hospital will be combined with the group at the Norwood Hospital.

MASSACHUSETTS LEGISLATIVE NOTES

Senate 321 which provides for an investigation of the desirability of the creation of a State Hospital for the treatment of infantile paralysis and arthritis has been favorably reported in the House.

Senate 51 which was designed to provide a division of the North Reading State Sanatorium for the treatment of cancer cases has been given leave to withdraw.

House 35, designed to provide for the annual registration of physicians, has been referred to the next annual session.

House 949, designed to abolish compulsory vaccination, has been given leave to withdraw.

MISCELLANY

THE APPOINTMENT OF DR. HANS ZINSSER

Dr. Hans Zinsser, professor of bacteriology and immunology at the Harvard Medical School since 1923, has been appointed Charles Wilder professor of bacteriology and immunology. He succeeds Professor Milton J. Rosenau.—*Science*, Vol. 83, No. 2151.

THE MASSACHUSETTS CENTRAL HEALTH COUNCIL

With no paid officers or executive, the Massachusetts Central Health Council has recently held an election and has appointed a legislative committee. This Council has worked to aid the passage of the medical education bill of this session and had much to do previously with the successful launching of the present Massachusetts State Health Commission, now engaged through its fourteen committees in a study of the health needs and laws of the Commonwealth. After the forthcoming report of this Commission in 1936, the Council hopes that its constituent health agencies will render important service in promoting indicated changes in health work procedure and to win legislation.

The elected officers are Miss Sophie C. Nelson, of the John Hancock Mutual Life Insurance Company, President; Dr. Gaylord W. Anderson, of the State Department of Public Health, Vice-President, and Arthur J. Strawson, of the Massachusetts Tuberculosis League, Secretary-Treasurer.

The constituent agencies of the Council, each represented by two administrative board members, are as follows: Dental Hygiene Council of Massachusetts, Massachusetts Public Health Association, Massachusetts Organization for Public Health Nursing, Massachusetts Department of Public Health, Massachusetts Medical Society, Massachusetts Department of Labor and Industries, Massachusetts Society for Mental Hygiene, Massachusetts Society for the Control of Cancer, Massachusetts State Nurses' Association, Massachusetts Tuberculosis League, Massachusetts Veterinary Association, Massachusetts Society for Social Hygiene, New England Heart Association, Metropolitan Chapter of the American Red Cross, New England District of the American Association of Hospital Social Workers and New England Health Education Association.

DR. H. W. SCHOENING NEW ASSISTANT CHIEF Of BUREAU OF ANIMAL INDUSTRY

Appointment of Dr. Harry W. Schoening, chief of the Pathological Division of the Bureau of Animal Industry, as an assistant chief of the Bureau, is announced by the U. S. Department of Agriculture. Dr. Schoening will continue as chief of the Pathological Division, but will devote time also to administrative duties dealing with general Bureau research in livestock diseases.

He was named chief of the Pathological Division in 1932 to fill the vacancy created by the death of his predecessor, Dr. J. S. Buckley.

Dr. Schoening is a native of Philadelphia, Pa., and holds a commission as a major in the Reserve Corps of the Army Veterinary Corps. He was a second lieutenant of the Army Veterinary Corps in 1918. He also is a member of the American Veterinary Medical Association, the Society of American Bacteriologists, the National Association of the Bureau of Animal Industry Veterinarians, and the Academy of Science.

SMOOTH OLIVE OIL RACKETEERS FOILED BY FEDERAL FOOD MEN

It was a good racket while it lasted—the "olive oil" racket with cheap tea seed oil substituted for the more expensive olive oil. But a Federal chemist has at last found a way to identify the tea seed oil, and the Food and Drug Administration has started legal action to confiscate thousands of gallons of the adulterated oil. The salad oil racketeers will now have to abandon tea seed oil as a source of illegitimate profits just as in the past they have had to abandon cottonseed oil, peanut oil, sesame oil, sunflower seed oil and others as adulterants of olive oil.

It is a long story with the racketeers shifting

from one cheat to another trying to keep ahead of the Government and the Government chemists working in their laboratories to devise methods of exposing the cheats, methods so conclusive that they would stand up in court and cause conviction of the cheaters. Time and again the laboratory men have caught up and the test tube has exposed one variety of adulteration, forcing the racketeers to a new dodge.—*Bulletin, U. S. Department of Agriculture.*

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR FEBRUARY, 1936

	Feb., 1936	Feb., 1935	5-Yr. Average*
Anterior poliomyelitis	2	2	2
Chickenpox	1220	1202	1135
Diphtheria	32	39	116
Dog bite	503	506	288
Epidemic cerebrospinal meningitis	23	2	5
German measles	424	1908	461
Gonorrhea	396	360	442
Lobar pneumonia	799	475	547
Measles	2477	2097	3095
Mumps	2213	290	638
Scarlet fever	1095	723	1354
Syphilis	426	379	339
Tuberculosis, pulmonary	214	316	279
Tuberculosis, other forms	28	38	34
Typhoid fever	9	1	8
Undulant fever	4	3	
Whooping cough	310	817	852

*Based on the figures for the preceding 5 years.

RARE DISEASES

Actinomycosis was reported from Boston, 1.

Anterior poliomyelitis was reported from Lowell, 1; Malden, 1; total, 2.

Anthrax was reported from Peabody, 1; Pittsfield, 1, total, 2.

Diphtheria was reported from Athol, 1; Boston, 12; Dracut, 1; Fall River, 3; Haverhill, 2; Lowell, 5; Malden, 1; Melrose, 1; North Attleboro, 1; Revere, 1; Somerville, 1; Tewksbury, 2; Worcester, 1; total, 32.

Encephalitis lethargica was reported from Townsend, 1.

Epidemic cerebrospinal meningitis was reported from Boston, 14; Lynn, 1; Newburyport, 1; Newton, 2; Rockland, 1; Spencer, 1; Webster, 1; Winchester, 1; Worcester, 1; total, 23.

Pellagra was reported from Nahant, 1; North Adams, 1; total, 2.

Septic sore throat was reported from Beverly, 1; Billerica, 1; Boston, 5; Chicopee, 1; Easton, 1; Fitchburg, 1; Gardner, 2; Haverhill, 1; Marshfield, 1; Milton, 1; Newton 1; Stoneham, 1; total, 17.

Trachoma was reported from Worcester, 2.

Trichinosis was reported from Agawam, 1; Cambridge, 1; total, 2.

Typhus fever was reported from Boston, 2; Petersham, 1; total, 3.

Undulant fever was reported from Beverly, 1; Norfolk, 1; Sheffield, 1; Weston, 1; total, 4.

Diphtheria was reported to a lower figure than for February, 1935, after a poor start in January due to a local outbreak.

Although most of the increase in reported epidemic cerebrospinal meningitis is due to outbreaks in two institutions, it would appear that the incidence in the State as a whole will be greater than usual throughout the year.

Typhoid fever shows as yet no decrease over last year's record low incidence.

The reported incidence of lobar pneumonia was higher than it has been for February since 1929.

Tuberculosis morbidity is running somewhat lower to date than in 1935.

Mumps continues to be reported in record breaking figures.

Scarlet fever continues to run higher than for the past two years.

Whooping cough had its lowest reported February incidence.

The incidence of anterior poliomyelitis, chickenpox, measles, German measles, and tuberculosis other forms was not remarkable.

COMMITTEE FOR THE STUDY OF SUICIDE

An organization to be known as the Committee for the Study of Suicide, Inc., was incorporated last December under the laws of the State of New York and began its activities early in January. The Committee may in time increase its present membership of ten to a total number of twenty. The Board of Directors and the officers of the new corporation are the following: Dr. Gerald R. Jameison, President; Mr. Marshall Field, Vice-President; Dr. Henry Alsop Riley, Treasurer; Dr. Gregory Zilboorg, Secretary and Director of Research; Miss Elisabeth G. Brockett, Dr. Franklin G. Ebaugh, Dr. Herman Nunberg, Dr. Dudley D. Schoenfeld, Dr. Bettina Warburg.

The Committee plans to undertake a comprehensive study of suicide as a social and psychological phenomenon. To achieve this the following general outline was adopted:

1. *Intramural studies* of individuals inclined to suicide in selected hospitals for mental diseases. These will embrace constitutional, neurological, psychiatric and psychoanalytic investigations of the phenomenon with special reference to therapy and prevention. This part of the study will include the investigation of suicidal trends or ideas of death emerging in organic deliria.

2. *Extramural studies* of ambulatory cases afflicted with suicidal trends or with obsessional wishes for their own death. These studies will be primarily therapeutic in nature, the cases to be treated in especially selected out-patient clinics and by qualified psychiatrists and psychoanalysts. Regular "control seminars" to follow and to supervise the course of the cases under treatment will be held

under the guidance of the Committee. The medical and neurological status of all cases will be a prerequisite of each case record.

3. *Social studies* of suicide will be undertaken along the following general lines. Various attempts at suicide will be followed up by experienced psychiatric social workers; all cases will be studied from the standpoint of social background and history and those who failed in their attempts or have recovered from injuries following a partially successful attempt (prolonged unconsciousness or physical illness) will be urged to submit to psychiatric and psychoanalytic treatment in the hands of the intra- or extramural therapeutic agencies which will be available to the Committee.

4. *Ethnological studies*, i.e., comprehensive investigation of suicide among primitive races, will be one of the first concerns of the Committee, for suicide is a rather frequent occurrence among many primitive races still extant and when studied may throw some light on suicide as a psychobiological phenomenon. It is planned that an expedition headed by a psychiatrically schooled anthropologist, a psychiatrist and a psychoanalyst should work for a time in a region such as the Melanesian Islands or the Gulf of Papua, and in the interior of the Mexican North West as well as among some of the North American Indian tribes. Further details of this plan will be elaborated.

5. *Historical studies* of suicide will be pursued systematically under the auspices of the Committee, so as to make available a scientific history of the phenomenon as a social and medicopsychological problem.

The Committee was organized under the guidance of its first chairman, the late Dr. Mortimer Williams Raynor, Medical Director of Bloomingdale Hospital, who died on October 5, 1935.

Dr. Henry E. Sigerist, Professor of the History of Medicine at Johns Hopkins University, and Dr. Edward Sapir, Professor of Anthropology at Yale University, are consultant members of the Committee. They will advise and guide in that part of the work which touches their respective fields.

The Executive Offices of the Committee are located at Room 1404, the Medical Arts Center, 57 West 57th Street, New York City, and will be in charge of an executive assistant.

CORRESPONDENCE

SENATE BILL 394

Board of Registration in Optometry
State House, Boston

March 16, 1936.

Editor, *New England Journal of Medicine*,

In your issue of March 12 there is an article calling attention to the danger inherent in Senate Bill 394. I do not question the sincerity of the writer, but his article shows a complete misunderstanding of the bill, even as it is printed. I would greatly appreciate the courtesy if you will publish this statement of fact:

The sponsors of this bill have no desire to encroach upon the privileges of the medical profession; in their desire to correct an abuse which has crept into the practice, by virtue of the exemption of application of the law to physicians. The language of the original bill was such that physicians would be subject to the rules of optometric procedure in an examination of the patient. We can see just cause for protest on the part of physicians to this regulation on principle alone, and since the matter was called to our attention, after consultation with some of the leading ophthalmologists of Boston, we have eliminated the objectionable phrase: "subject to rules or regulations governing the practice of optometry" and inserted in its place "subject to rules pertaining to advertising". This phrase is necessary to take care of the situation where a few unethical physicians are prostituting the profession by the use of their certificates in the practice conducted by these unscrupulous commercial concerns; and surely no ethical physician would protest at the regulation of bait advertising by anyone, be he physician, optometrist or optician. The bill in every other particular does not affect physicians; they are exempt from the provisions of the bill in the original law and in these proposed amendments; they are also exempt from all of the provisions of the law, save this one pertaining to advertising. I would respectfully refer any physician who questions the accuracy of the above statements to consult with some of the leaders of the New England Ophthalmological Section for verification of these statements. There will be a hearing on March 19, and we would welcome the attendance of any physician to hear our presentation of the case with an open mind, and thus be reassured of our honesty of purpose, and of the fact that no ethical physician has reason to protest the enactment of his legislation. Thanking you for the courtesy of your columns, I am

Very truly yours,

JOHN E. CORBETT, *Chairman*,
Massachusetts Board of Registration
in Optometry.

EDITORIAL NOTE: An agreement has been reached at a meeting of physicians and optometrists for an amendment to this bill which is satisfactory to both parties.

COEXISTENCE OF APPENDICITIS AND MEASLES

March 17, 1936.

Editor, *New England Journal of Medicine*,

The current increase in the normal average number of cases of measles reported from certain communities in Eastern Massachusetts together with a recent experience prompted me to call attention again to the coexistence of acute appendicitis and measles.

In this Journal for April 12, 1934, a letter was published in the correspondence section in which I reported several personal experiences with measles and appendicitis. A month after this letter appeared I saw another child, aged five, who was taken ill on May 7, 1934, with measles. On the evening of May 10 she vomited. On May 11 she seemed more ill than she had been during the preceding three days. On May 12 she complained of abdominal pain and there was more vomiting. On May 13, the day on which I saw her, she again complained of abdominal pain. Her examination at that time revealed the presence of an appendix abscess. She was operated upon that day and a retrocecal perforated appendix with abscess formation was found. The appendix was removed and the abscess drained. She made a gratifying convalescence.

At about this same time a five-year-old boy was seen by one of my friends. This child complained of abdominal pain and had been vomiting for several days. His examination was suggestive but not diagnostic of appendicitis. There was a leucopenia, and Koplik spots were seen on his mucous membranes. He was observed for twenty-four hours during which time he developed signs of peritonitis. He was operated upon and the peritonitis found to be of appendiceal origin. This patient died.

On March 14, 1936, I saw a girl of eight who was taken ill early in the morning with vomiting. She vomited throughout the morning and the early afternoon. There was no complaint of abdominal pain. When seen by her physician at one-thirty P.M. on March 14 her temperature was 99° and the physical examination was considered negative except for slight redness of the throat and slight injection of one ear drum. At four-thirty P.M. she complained of abdominal pain for the first time. At six-thirty P.M. she was seen by her physician who made a diagnosis of acute appendicitis. Operation was performed at nine-thirty P.M. and an acutely inflamed appendix removed. The lumen of the appendix was distended with pus. Following operation on March 14 her convalescence has been satisfactory except for the presence of slight fever. She vomited once the evening of March 15 and once the afternoon of March 16. This afternoon a diagnosis of measles was established.

As pointed out in the letter of 1934 this experience has been noted by others, chiefly in foreign literature. A review of the subject with six reported cases appears in the *Archives of Pediatrics* for January, 1933.

This letter is written in the hope that practitioners seeing children with measles who present signs suggestive of appendicitis will give consideration to the latter as a probable diagnosis, as in the eight cases of which I have personal knowledge all but one had perforated before operation, with the result that two of the eight children died.

HENRY W. HUDSON, JR.

1101 Beacon Street,
Brookline, Mass.

SURGICAL OPERATION FOR HIGH BLOOD PRESSURE

March 16, 1936.

Editor, *New England Journal of Medicine*,

Your editorial of March 12, "Surgical Operation for High Blood Pressure," is timely and justifiably conservative in regard to this new therapeutic approach to a very common disease; your plea for more experimental work and less surgical trial is, in general, appropriate. However, the employment of certain surgical procedures in my opinion is justified in carefully selected cases after all the resources of a thorough medical régime including psychotherapy have been exhausted.

To date the surgical procedure of choice is dorsal sympathectomy or possibly as suggested by the Mayo Clinic[1] splanchnic resection and partial adrenalectomy. Laminectomy and anterior root section appear to carry too great a mortality and too great a risk of postoperative complications. Some experimental work[2] has been interpreted as demonstrating that surgery of the sympathetic nerves will not be effective. The work of others[1, 3] and the experience of Dr. R. H. Smithwick and myself, as yet unreported, nevertheless show that operative procedures are effective in lowering the blood pressure in certain cases during postoperative periods of months up to slightly over two years.

The selection of cases is most important. After the menopause in women and over forty-five years of age in men, essential hypertension pursues a long and benign course lasting ten to twenty years, during a large part of which the disease is either asymptomatic or symptoms are favorably influenced by simple measures. Essential hypertension in young males, or vasomotor instability with transient rises in blood pressure often spontaneously subsides or is readily controlled. On the other hand, rarely in young males, but more often in young females before the menopause, in certain constitutional types, observation reveals an intensity, a rapidity of progress, and a failure to respond to treatment which suggests the approach or onset of the malignant phase of essential hypertension. If abnormal catamenia, sterility, or toxemia of pregnancy occurs in the anamnesis one is more inclined to a grave prognosis. If such a patient under careful treatment shows progress of the disease I believe the use of a relatively safe surgical procedure with a reasonable prospect of success may be offered *with full explanation* to the patient.

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3. Page, I. H., and Heuer, G. J.: Surgical treatment of essential hypertension. J. Clin. Investigation **14**: 22 (Jan.) 1935.

ROBERT S. PALMER, M.D.

330 Dartmouth Street,
Boston.

BIOLOGICAL PROBLEMS OF STERILIZATION

To the Editor of *The New York Times**:

Without entering into the controversy on sterilization in any direct sense, let me state briefly the fundamental facts uncovered by the committee of the American Neurological Association and its conclusions and recommendations.

We find the claims of most eugenists as to the incidence of mental disease and mental defect unwarranted. There is no evidence of an actual increase. The entire biology of the situation is opposed to any real increase. Thus, it is not true that the feeble-minded have large families or are more prolific than the general population, nor is this true of the insane.

All the facts indicate quite clearly that there is a low marriage rate, a relatively low birth rate, a high death rate and even a high divorce rate. This is not only our opinion but also that of the British commission. That the biological setting is directly against any increase of the incidence of mental disease and mental defect is brought out by an actual analysis made not only for the United States but the world by Dr. Ellen Winston. The only present increase in mental disease in any of the cultural countries relates to the incidence of the senile mental diseases, and this is undoubtedly due, first, to the greater age of the population, accounted for by the lower birth and death rate, and, second, to the fact that as hospitals grow better, people are more ready to send their aged sufferers to institutions for mental disease.

"MYTHICAL MONSTROSITIES"

The Nams, Kallikaks, Jukeses and similar families are, in the opinion of the British commission and of the American committee, more or less mythical monstrosities which are probably largely social in origin.

Certain physical and mental diseases may be stated to have a hereditary basis. A few rare bodily diseases, such as Huntington's chorea, hereditary blindness and deafness, are unquestionably of genetic origin. The main mental diseases with which eugenic legislation is concerned are manic-depressive psychosis, dementia praecox, epilepsy, feeble-mindedness and, by a stretch of the term "mental disease," criminality.

While there is a hereditary basis in the case of manic-depressive psychosis and dementia praecox, it can safely be stated that the mechanism of heredity is entirely unknown. The inheritance of these conditions is not classically Mendelian; probably some environmental factor is at work as well as the hereditary one. The attempt to prevent these diseases by sterilizing those who themselves are not sick would be futile. Furthermore, sterilization would not greatly reduce their incidence. In a certain sense, dementia praecox sterilizes itself. Those who suffer from it have a low marriage and birth rate and tend to early incarceration in hospitals.

Of feeble-mindedness it can be directly stated that a large proportion is of hereditary origin. Yet the mechanism of inheritance is debatable and does not conform to Mendelian ratios or any modification thereof.

THE CARE OF EPILEPSY

Epilepsy can be, practically speaking, eliminated from consideration as hereditary. At the last meeting of the international neurological bodies in London the statement was made directly by several of the readers that environmental difficulties could be assumed to create the disorder.

As to criminality, none of the important geneticists believe that sterilization would have any effect; that, whatever "constitution" is involved, criminality is so linked up with culture, economics, tradition and the pressure of the complex social environment as to preclude sterilization as a defensive weapon.

Our committee takes note of the fact that considerable genius is associated with mental disease; that especially is the manic-depressive temperament closely related to superior ability, and that any sterilization procedures which operate blindly and without taking into account the total assets of the personality may do more harm than good. It becomes quite obvious from a study of literature that much genius would have been lost if drastic sterilization laws had been enacted in times past.

This committee, therefore, recommended in essence as follows:

1. That on the American scene compulsory sterilization is futile and only voluntary selective sterilization laws can be recommended, this to apply to inmates not only of State institutions but of private institutions and to individuals in the community at large. This might reasonably apply to those cases of feeble-mindedness of definite hereditary origin, but even here the assets of the personality in other directions might well be considered.

SELECTION URGED

2. That in the case of manic-depressive psychosis there should be a very careful weighing of the individual case and of the total personality assets and liabilities. In other words, we recommend strongly selective sterilization as the only legitimate legislation here to be considered.

3. This applies, although in lesser degree, to dementia praecox.

4. In so far as epilepsy is concerned, we are strongly opposed to any sterilization on the basis of heredity, although there may be a reason why an individual who has epilepsy should be voluntarily sterilized because of other difficulties he encounters. The committee is opposed to sterilization for social reasons or social difficulties, largely because of the danger of forging a weapon which would be used by the unscrupulous for punitive and prejudicial purposes.

5. In so far as crime is concerned, this committee is wholly opposed to sterilization, believing that it is only a form of punishment, a sort of dodging

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of the issue, and a shifting of the responsibility from society to the germ plasm.

The crying need of eugenics, as this committee sees it, is not legislation but real research. There have been no researches which fully merit the term scientific. The difficulties are great and they require a large organization with a systematic study, especially control studies, carried over a period of fifteen to twenty-five years. While we recommend a limited legislative program, our main recommendation lies in the direction of more fundamental, more thorough and more impartial research.

ABRAHAM MYERSON, M.D.,
Chairman, American Neurological Association Committee for the Investigation of Sterilization.

Boston, Mass., March 10, 1936.

RECENT DEATH

McALLISTER — FREDERICK DANFORTH MCALLISTER, M.D., of Methuen, with an office at 301 Essex Street, Lawrence, died at the Lawrence General Hospital, March 17, 1936.

Dr. McAllister, the son of Dr. John D. McAllister, was born in Lawrence in 1872, attended Amherst College and graduated from the Harvard Medical School in 1898. His interneship was served at the Worcester City Hospital.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association and had been prominent as a surgeon and consultant.

His widow, a daughter, two sisters and a brother survive him.

NOTICES

THOMAS WILLIAM SALMON MEMORIAL LECTURES

The Salmon Committee on Psychiatry and Mental Hygiene invites the medical profession and their friends to the Fourth Series of Thomas William Salmon Memorial Lectures to be given by Samuel T. Orton, M.D., Friday evenings (8:30), April 10, 17, 24, 1936, at the New York Academy of Medicine, 2 East 103rd Street, New York City.

Developmental Disorders of the Language Faculty and Their Psychiatric Import

Lecture I. Language Losses in the Adult as the Key to the Developmental Disorders in Children (April 10).

A discussion of the physiological background of the language faculty as revealed in the aphasias. The problem of unilateral cerebral dominance. Reports of studies indicating that all degrees of intermixture occur between right- and leftsidedness. Such intergrading between the hemispheres is suggested as the background for many language disorders in children.

Lecture II. The Syndromes of Disorder in the Development of Language (April 17).

Six syndromes are discussed from the point of view of their symptomatology: Reading Disability (strephosymbolia), developmental word deafness, congenital apraxia, motor speech delay, writing disability, and stuttering.

Lecture III. Treatment and Psychiatric Interpretation (April 24).

A brief review of the general principles of treatment of the various syndromes, together with a discussion of the relation of these conditions to emotional and mental development.

BOSTON DISPENSARY

25 Bennet Street, Boston
Medical Conference Program
9-10 A.M., April, 1936

Wednesday, April 1 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, April 2 — Endocrine Clinic. Dr. C. H. Lawrence.

Friday, April 3—Lobectomy and Pneumonectomy for Bronchiectasis and Tumors of the Lung. Dr. Richard H. Overholt.

Saturday, April 4 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, April 7—Diseases and Injuries to the Hip Joint. Dr. John D. Adams.

Wednesday, April 8—Recognition of the Early Psychoses; Their Differentiation from Neuroses. (Continued.) Dr. A. Warren Stearns.

Thursday, April 9—Development of Method in Psychopathology. Prof. Elton Mayo.

Friday, April 10—Rheumatic Fever. Dr. Clifford L. Derick.

Saturday, April 11 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, April 14—Etiological Factors in Anemia. Dr. William Dameshek.

Wednesday, April 15—Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, April 16 — Seasonal Hay Fever. Dr. Joseph Kaplan.

Friday, April 17—Certain Aspects of the Thyroid. Dr. David Rapport.

Saturday, April 18 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, April 21—X-Ray Demonstration. Dr. Alice Ettinger.

Wednesday, April 22—Pericarditis. Prof. G. Klemperer.

Thursday, April 23—Social Service Case Presentation. Miss E. R. Canterbury.

Friday, April 24—Autonomic Pharmacological Experiments on the Human Being. Dr. Abraham Myerson.

Saturday, April 25 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, April 28—Pediatric Case Presentation. Dr. Francis McDonald.

Wednesday, April 29—Hospital Case Presentation. Dr. S. J. Thannhauser.

Thursday, April 30—Some Practical Aspects of Tuberculosis. Dr. C. H. Whitehurst.

REPORTS AND NOTICES OF MEETINGS

FORTY YEARS OF X-RAY

On Thursday, January 23, 1936, the fortieth anniversary of Professor W. K. Roentgen's discovery of the x-ray was commemorated in the Moseley Memorial Building of the Massachusetts General Hospital. Dr. W. B. Breed presided, and commented on the modesty and sincerity of Professor Roentgen, and on his enormous capacity for work.

Dr. Francis T. Hunter reviewed the main events of Roentgen's life. He was born in 1845, and it is not definitely known whether he was a citizen of Holland or Germany. He was not particularly brilliant, and because he was refused entrance to the gymnasium, he gained his early education in the Polytechnic School of Zurich. He received his doctorate of philosophy from the University of Würzburg in 1871, but could not gain academic promotion in that school because of his unfamiliarity with Latin.

He moved to the new University of Strassburg in 1876, where he was an instructor in physics, and performed important researches on gun metals. Because of his excellent work he became well known academically, and was offered the chair in physics at the University of Utrecht, a school which had refused him entrance as a student. He declined this appointment, and in 1888 accepted the professorship of physics at the University of Würzburg, where he had previously been denied advancement because of his lack of knowledge of Latin. In 1895 he began his experiments on cathode rays, which were to lead to his discovery of the x-ray.

On December 20, 1895, his paper Eine Neue Art vor Strahlen appeared in the Setzungsberichte der Würzburger Physik-medic. Gesellschaft. Even before this periodical was distributed, reprints had been prepared, and news of his discovery had reached the press, and been widely publicized over the whole of the civilized world. It was not until January 23, 1896, that he delivered his paper before a large audience of academics and high government officials.

In 1900 he became professor of physics at Munich, and was awarded the Nobel Prize in physics in 1901. He received some fifty honorary degrees from various universities all over the world, before his death in Munich on February 10, 1925, at the age of seventy-eight years.

The discovery of the x-ray must be considered with the discovery of ether anesthesia, and the bacterial etiology of disease as one of the three greatest contributions to the medical knowledge of the world to be made in the nineteenth century.

Dr. E. A. Codman spoke on Reminiscences of Early X-Ray Work in Boston. The announcement of the x-ray and its properties aroused a vast amount of skepticism, and the first review of the work in the *Boston Medical and Surgical Journal* of February 13, 1896, was extremely conservative in its comment. Dr. Codman published an article in this Journal March 20, 1896, with reproductions of x-ray plates of a full-term fetus, showing the varying calcification of different bones, and the ossification centers. Four months elapsed after Roentgen's announcement of his discovery before practical application of the rays were made in Boston, when on April 13, 1896, Dr. Codman took an x-ray photograph of a hand in which a shoemaker's brad was imbedded.

A "double focus" roentgen tube was secured from England in May, 1896, which greatly aided in photography of the deeper structures, and studies of the heart and lungs were made by Dr. Francis Williams in October of that year. Investigations of the effects of x-rays on tissues were first made in September by Dr. Seabury Allen, and Dr. J. C. White first reported x-ray burns in December of 1896. The story of the earliest use of the x-ray in Boston, the first description of x-ray dermatitis, the first report of the effect of radiation on the tissues are all contained in the 1896 volume of the *Boston Medical and Surgical Journal.*

Mr. Joseph Goodsoe, who worked in the x-ray department of the Massachusetts General Hospital from 1896 to 1909, spoke of his experiences with the various forms of apparatus in use at that time, and paid tribute to Dr. Walter Dodd, one of the first martyrs to the advancement of x-ray.

Dr. George W. Holmes, roentgenologist at the Massachusetts General Hospital since 1911, spoke on The Present Status of Radiology in the Treatment of Cancer. Dr. Holmes reported a case of carcinoma of the lip, which he treated in 1910 with fractional doses of radiation over a considerable period of time. This patient was followed for ten years, with no signs of recurrence. In the application of radiation therapy it is important to consider the age of the patient, the size, location, and malignancy of the lesion to be treated. The problem in treating lesions lying beneath the surface of the body is to give these lesions the greatest possible dosage with as little damage as possible to the overlying tissues. The dosage to the surface is always greater than the depth dose, with increasing voltage there is an increase in the depth dose, with increasing distance between the patient and source of the rays, the radiation received by the deeper tissues approaches the amount received at the surface. The upper limit of increased penetration due to voltage increase is reached at about 500 KV., and 80 cm. is the maximum distance at which treatment is practical.

When extremely high voltages are employed in treatment, the character of ionization within the tissues is changed, and as a result the therapeutic effects may be different from those obtained with lower voltages. The data at present available do

not indicate that these results will be superior, however.

The degree of malignancy is of importance in determining the value of radiation therapy, in a given case. Many highly malignant tumors are easily destroyed with but slight injury to surrounding tissues. They are prone to metastasize early, however, and are seldom cured. Tumors of low malignancy are more radioresistant, and effective radiation does more damage to surrounding tissues, but due to the relatively late occurrence of metastases, results obtained in treating such lesions are often better than those in the more radiosensitive group.

Clinical experience is of the utmost value in applying radiation therapy. No tumor should be treated with massive dosage until absolutely proved to be malignant. Consultation service is considered necessary in every case, and only after the diagnosis has been established, the consultant has expressed his opinion, and a definite course of procedure has been adopted, should therapy be instituted. A plan of treatment once adopted should not be varied without further consultation, and thorough consideration. The tumor clinic with a staff of physicians of several different specialties each seeing the case is of distinct value.

It must be remembered that the tissues of children and youthful persons are more easily injured by radiation than those of elderly persons, and that growth processes may be permanently arrested by radiation.

Treatment may be undertaken with the hope of curing the patient, or as a means of palliation, that is relief of distressing symptoms. Treatment should be refused in cases with incurable disease which do not suffer disturbing symptoms, since radiation may only make such patients more uncomfortable, and is unjustified.

The question of preoperative radiation treatment with the hope of making more cases operable has recently been revived. If a case is inoperable, radiation will not make it operable. Cases which are inoperable because of their inaccessibility to surgical approach may be benefited and sometimes cured by radiation. Occasionally a case can be made "safer" for operation, e.g., large kidney tumors may be reduced in size, or the danger of rupture and peritoneal implantation of certain ovarian tumors may be minimized.

Postoperative radiation treatment is not logical. If the lesion cannot be removed by surgical means it should not be operated upon but treated with radiation alone, and if it can be removed completely there is no reason for postoperative radiation. If an attempt at complete removal fails and tumor tissue is left behind, treatment should be carried out as though there had been no operation.

Preradiation care is of importance. Local treatment should eradicate infectious processes, especially in the mouth. Bad teeth should be removed before radiation is begun, and not after, since radiated tissue is peculiarly susceptible to serious infection. The general condition of the patient should be improved as much as possible. Food and fluid intake should be adequate, and anemia should be alleviated. Necessary postoperative radiation should not be instituted until sufficient time has elapsed to allow complete recovery from the ill-effects of operation. During treatment a high carbohydrate diet, and sufficient fluid intake, should be maintained, and every effort made to prevent the psychic suggestion of possible radiation sickness. If, in the treatment of the lesion, epilation is produced, skin ointments should be used only with caution, since they prevent the evaporation of perspiration, elevate the skin temperature, and aggravate the reaction. If heavy treatment is directed to tumors in the upper respiratory passages, facilities for the performance of tracheotomy must be at hand.

There are three types of "roentgen sickness": The first is of psychic origin, and is often eliminated by the substitution of radium for x-ray, or vice versa, or by referring the patient to another laboratory. The second type is due to "intoxication" from tissue breakdown, and can be treated in the manner employed in the treatment of other "toxic" conditions. "True" roentgen sickness, which is of unknown etiology, has many features suggestive of liver dysfunction. Treatment of this condition with a high carbohydrate diet definitely aids some cases.

The belief that high voltage is more likely to cause radiation sickness than low voltage is false. The development of roentgen sickness is independent of the voltage used for the treatment, and depends upon the total area treated, the location of this area, and the dose administered.

Dr. Holmes believes that advance in the field of roentgen therapy will be made through variations in the duration of treatment, and fractionation of dosages, rather than by use of extremely high voltages.

THE BOSTON PATHOLOGICAL SOCIETY

The stated meeting of the Boston Pathological Society was held in the Pathology Laboratories of the Infants' and Children's Hospitals on Wednesday evening, February 19. The first part of the evening was devoted to an exhibition and demonstration of interesting pathological specimens obtained at the Infants' and Children's Hospitals and from members of the society.

Following this, the meeting was formally called to order by Dr. Monroe J. Schlesinger, President, and a series of papers was read. Dr. Robert Fienberg of the Pondville Hospital in Wrentham discussed his recent work in determining the actual mechanism of the Smith-Dietrich lipoid stain, as well as further studies he is conducting in staining various members of the lecithin-phosphatide groups. Then there were four papers on various unusual and bizarre types of tumors. The first presentation was by Dr. B. Earl Clarke of the Rhode Island Hospital, Providence, on "Retinal Tumors in Tuberous Sclerosis". Dr. Clarke reviewed the literature and showed lantern slides demonstrating the microscopic ap-

pearance of a case of retinal tumor in this disease. The nature of the tumor cell was discussed in some detail but its exact origin was not definitely determined. Perhaps it is related to the primitive cells from which the rods and cones originate.

Dr. Paul Harris of the New England Deaconess Hospital reported a case of onkocytoma of the parotid gland. In discussing this particular tumor Dr. Harris pointed out that so-called onkocytes, which are large, granular, swollen cells, occur in the mucous and serous glands in the mouth in older persons only. While the exact nature of the cell is not known, it may represent the result of the process of so-called dedifferentiation. Rare though the cells are, it is even more rare for them to become malignant.

Dr. John Egoville presented a very interesting case of pheochromocytoma of the adrenal gland, from the Rhode Island Hospital. The patient, a young woman, had had paroxysmal symptoms of hypersecretion of adrenalin, and a tumor of the adrenal medulla was found and removed. Unfortunately, the patient died following operation. The last case was a presentation by Dr. Edward Bosworth of the Rhode Island Hospital, in which there were primary mesothelioma cells studded throughout the pleural, pericardial, and peritoneal cavities.

Following the scientific meeting, members and guests adjourned for refreshments.

CARNEY HOSPITAL CLINICAL MEETING

The next meeting will be held Friday evening, April 3, at 8:30.

Subject: Chronic Nephritis. Dr. James P. O'Hare.

Physicians and medical students are invited.

FAULKNER HOSPITAL CLINICAL MEETING

The next meeting will be held on Thursday, April 2, at 5:00 P.M.

In addition to the usual clinical pathological conference Dr. Frederic J. Cotton and Dr. Henry C. Marble will talk on Reconstruction Surgery—Joint, Bone and Tendon.

All physicians are invited.

THE NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the society will be held in Hotel Kenmore, Boston, Tuesday evening, March 31, 1936. Tel. Ken. 2770.

Business Meeting: 7:30 P.M. Chairmen of committees are asked to bring in reports.

Communication: 8:15 P.M.

Cauterization of the Cervix Uteri Using Various Electrical Methods. Illustrated. Dr. Benedict F. Boland.

Discussion.

Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*
1236 Beacon Street,
Brookline, Mass.

BOSTON HOSPITAL COUNCIL

The first Annual Meeting of the Hospital Council of Boston will be held in the Lower Amphitheatre of the Out-Patient Department of the Massachusetts General Hospital on Monday, April 6, 1936, at 4:30 o'clock.

The business to come before the meeting is:

1. Election of officers.
2. Adoption of proposed amendments to the Constitution and By-laws, a copy of which is presented below.
3. Report of the Treasurer.
4. Report of the President of special activities during the past year.
5. Dr. Wilinsky will report on his personal impressions of the Medical Economic Security Council of Washington.

Since this is the first opportunity the Officers and Executive Committee have had to make a report of their activities to the entire Corporation, it is hoped that all members will make a special effort to be present.

J. B. HOWLAND, M.D., *President.*

PROPOSED AMENDMENTS TO THE CONSTITUTION AND BY-LAWS OF THE HOSPITAL COUNCIL OF BOSTON, TO BE ACTED UPON AT THE ANNUAL MEETING ON MONDAY, APRIL 6, 1936

Article VI. Elections, shall be amended as to read:

Article VI. Elections:

Section 1. (Amendment.) The officers and members of the Executive Committee to be elected at the Annual Meeting shall be nominated by a Nominating Committee appointed by the President one month before the Annual Meeting, but at the Annual Meeting nominations may also be made.

Section 2. (Amendment.) All vacancies for office shall be filled at the Annual Meeting; election to be by ballot unless otherwise ordered.

Section 3. The representatives of the organizations in the Council membership shall be elected or otherwise designated annually by their respective organizations, and notification of their selection sent to the Secretary of the Council at least seven days prior to its annual meeting.

Section 4. (Amendment.) In case a vacancy occurs in any of the offices or in the membership of the Executive Committee, the Executive Committee shall by ballot elect a person to fill such vacancy who shall hold office until the next Annual Meeting when such vacancy shall be permanently filled at the annual elections.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 30, 1936

Tuesday, March 31—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Pediatric Case Presentation. Dr. Francis McDonald.

*12 M. Boston Dispensary, luncheon meeting of the Clinical Staff. Auditorium, second floor, Dispensary Building.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

7:30 P.M. Norfolk District Medical Society, Hotel Kenmore, Boston.

Wednesday, April 1—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 M. Clinico-Pathological Conference. Children's Hospital.

Thursday, April 2—

*8:30-9:30 A.M. Clinic, Surgical Staff of the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Endocrine Clinic. Dr. C. H. Lawrence.

5 P.M. Faulkner Hospital Clinical Meeting.

Friday, April 3—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Lobectomy and Pneumonectomy for Bronchiectasis and Tumors of the Lung. Dr. Richard H. Overholt.

*8:30 P.M. Carney Hospital, Clinical Meeting.

Saturday, April 4—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

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*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

March 26—Medical Clinic, Peter Bent Brigham Hospital, 3:30 P.M.

March 26—Massachusetts General Hospital, Clinical Meeting, 8:15 P.M.

March 30—Springfield Medical Association, 8:30 P.M. at the rooms of the Springfield Academy of Medicine, 20 Maple Street. The Development of Surgical Practice in Springfield. Dr. John M. Birnie.

March 31—Boston Dispensary Clinical Staff Meeting, at 12 M.

April 1—Wachusett Medical Improvement Society. Holden District Hospital. Dinner 6:30 P.M. Business session and scientific program, 7:30 P.M.

April 1-30—Boston Dispensary, Medical Conference Program. See page 660.

April 2—Faulkner Hospital Clinical Meeting. See page 663.

April 3—Carney Hospital Clinical Meeting. See page 663.

April 6—Boston Hospital Council. See page 663.

April 8—Joint Meeting of the Massachusetts Tuberculosis League and the Hampden County Tuberculosis and Health Association. See "An address by Dr. Kendall Emerson." Page 498, issue of March 5.

April 10—William Harvey Society. Beth Israel Hospital. 8 P.M.

April 10, 17, 24—Thomas William Salmon Memorial Lectures. See page 660.

April 20-24—A Postgraduate Institute in Philadelphia. See page 497, issue of March 5.

May 1, 2, 3, and 4—The American Association on Mental Deficiency. See page 610, issue of March 19.

May 12-16—The International Congress of Physical Medicine. See page 443, issue of February 27.

June 15-19—The Executive Board of the Catholic Hospital Association will meet at the Fifth Regiment Armory, Baltimore, Md.

June 16-July 28—Summer Course in Bacteriology. See page 385, issue of February 20.

September, 1936—First International Conference on Fever Therapy. See page 1325, issue of December 26, 1935.

September 7-10—International Union against Tuberculosis. See page 554, issue of March 12.

October 19-23—Clinical Congress of the American College of Surgeons. See page 180, issue of January 23.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

April 1—Wednesday. Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Richard H. Overholt of the Lahey Clinic. Subject: Chest Surgery.

May 7—Thursday. Censors' Meeting.

May 13—Wednesday. Annual Meeting. Salem Country Club. Dinner at 7 P.M. Speaker: Dr. Paul White. Subject to be announced later.

R. E. STONE, M.D., Secretary.
88 Lothrop Boulevard, Beverly.

FRANKLIN DISTRICT MEDICAL SOCIETY

May 12—Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

May 6—Bear Hill Golf Club, Stoneham, at 12:15 P.M.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

March 31—See page 663.

May—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D., Secretary.
1236 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

April 16—Brockton Hospital.
May 21—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.
167 Newbury Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

April 29—Annual Meeting at the Boston Medical Library. The Treatment of Septicaemia, Dr. Champ Lyons. The Pleurality of Scarlatinal Streptococcus Toxin, Dr. Sanford B. Hooker. Discussion: Dr. Hans Zinsser.

The medical profession is cordially invited to attend this meeting.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

April 8—Wednesday evening. Hahnemann Hospital, Worcester, Mass. Dinner and scientific program.

May 13—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced in an April issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOK REVIEW

Human Pathology. A Textbook. Howard T. Karsner. Fourth Edition, Revised. 1013 pp. Philadelphia and London: J. B. Lippincott Company.

The fourth edition of Dr. Karsner's textbook maintains the standard of excellence set in the first. The well-selected and up-to-date references are a useful feature of each chapter. The newer knowledge of various phases of the subject is well incorporated, particularly in the sections dealing with the ductless glands, the nervous system, and the hematopoietic system. The section on the general pathology of tumors has been very extensively revised and is one of the best discussions that it has been the good fortune of the reviewer to read. A number of illustrations, particularly of microscopic sections, are line and stipple drawings, inferior to good photomicrographs. The drawings of gross specimens, on the other hand, maintain an excellent standard. The division of the book into chapters in general pathology as compared with special pathology is a satisfactory one, which fits in well with most medical school courses.